Acute renal vein thrombus and renal atrophy following shock wave lithotripsy: a unique complication

Matthew C. Ferroni, MD, Robert M. Turner II, MD, Michelle J. Semins, MD
Department of Urology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, USA

FERRONI MC, TURNER RM II, SEMINS MJ. Acute renal vein thrombus and renal atrophy following shock wave lithotripsy: a unique complication. *Can J Urol* 2014;21(1):7151-7153.

Serious complications of shock wave lithotripsy (SWL) are rare, but can have significant long term effects. We present a case of acute renal vein thrombus following SWL leading to subsequent renal atrophy and loss of renal function. To our knowledge this is a newly reported complication of SWL.

Key Words: shock wave lithotripsy, renal vein thrombus, nephrolithiasis

Introduction

Following its initial description in the early 1980s, shock wave lithotripsy (SWL) gained rapid acceptance and remains the most commonly utilized treatment modality for renal and proximal ureteral calculi < 20 mm in size.[1,2] The popularized use of SWL is due to its ease of performance and noninvasive nature. Complications are related mostly to residual stone fragments, infection, and traumatic effects to the renal parenchyma and adjacent tissues. Energy transmitted outside the focal point (F2) of the lithotripter may result in endothelial damage to the renal microvasculature with resultant tissue hypoxia.[3,4] Macrovascular complications are a rare event and have included rupture of abdominal aortic aneurysm, pseudoaneurysm, arterial stenosis, arteriovenous fistula, and venous thrombosis.[5] Herein we present a unique case of acute renal vein thrombosis following SWL for an 8 mm renal stone leading to significant renal functional loss within 3 months of treatment.

Accepted for publication December 2013

Address correspondence to Dr. Matthew C. Ferroni, Department of Urology, University of Pittsburgh School of Medicine, 3471 Fifth Ave, Ste 700, Pittsburgh, PA 15213 USA

Case report

A 47-year-old obese female, with a past medical history significant only for hypertension managed without pharmacotherapy, initially presented to an outside institution with acute-onset right flank pain, nausea, and vomiting. A non-contrast computed tomography (CT) scan revealed an 8 mm stone at the right ureteropelvic junction and mild hydronephrosis, Figure 1. One week later, SWL was performed; a

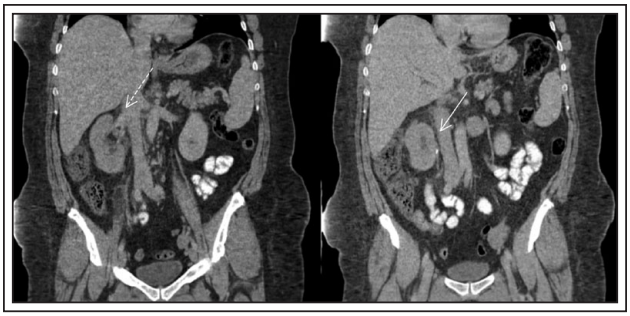

Figure 1. Non-contrast CT abdomen and pelvis showing an 8 mm proximal right ureteral stone (solid arrow) in relation to the right renal vein (dotted arrow) with early branching.

total of 3000 shocks were delivered at 24 kV using an HMT LithoTron table top unit under sedation. There was radiographic evidence of complete stone comminution. Five days following the procedure, she represented with shortness of breath and pleuritic chest pain. A chest CT scan demonstrated bilateral segmental and sub-segmental pulmonary emboli. Concurrent abdominal CT raised concern for a right renal vein thrombosis. An intravenous heparin infusion was administered, and she was transferred to our institution for further evaluation and management. Magnetic resonance imaging (MRI) confirmed a mildly enlarged right kidney with a non-enhancing thrombus in the right renal vein on T2-weighted images, Figure 2. A Doppler ultrasound of her lower extremities revealed no evidence of thrombus. Her past medical history was thoroughly reviewed which was negative for any previous episodes of thrombosis, and she denied any history of pharmacologic anticoagulants. Her family history was significant for a pulmonary embolism in her father. She subsequently underwent an extensive hyper-coagulation workup by hematology which was completely negative. She was maintained on systemic anticoagulation without event for 6 months. Three months post-SWL, repeat CT scan demonstrated complete resolution of the renal vein thrombus and heterogeneous enhancement of the right kidney with diffuse cortical loss, Figure 3a. Her serum creatinine remained within normal limits.

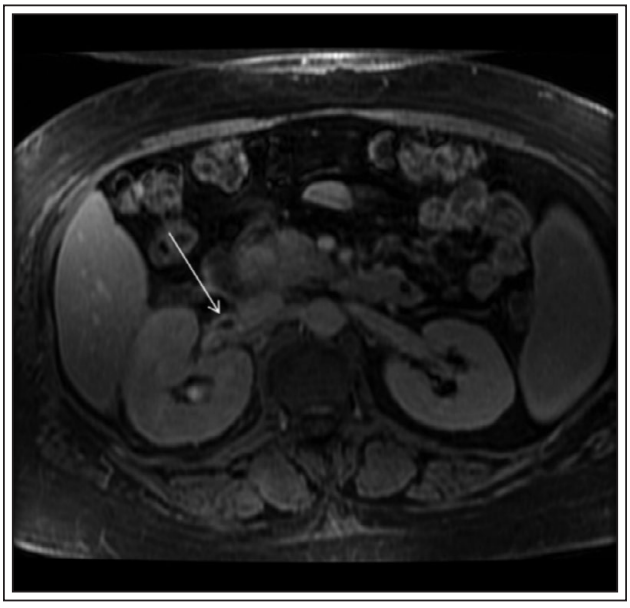

Figure 2. T2-Weighted MRI showing mild enlargement of right kidney and non-enhancing thrombus of right renal vein (arrow) without extension into IVC.

At 8 months post-SWL, the patient re-presented to the emergency department with acute left sided flank pain and was found to have an obstructing 4 mm proximal left ureteral stone. Her serum creatinine was 5.1 mg/dL indicating an obstruction of a functionally solitary left kidney. CT demonstrated progressive right renal atrophy with cortical lobulation and significant parenchymal loss. Her left ureteral stone was successfully treated with emergent stent to relieve obstruction followed by ureteroscopy and laser lithotripsy 2 weeks later. Her renal function, however, has remained chronically impaired with an estimated GFR ranging from 34 to 44 mL/min/1.73m^2 based on the CKD-EPI Creatinine Equation. A recent CT was obtained at 40 months post-SWL, showing continued lobulation and atrophy of her affected kidney, Figure 3b. She is followed closely in our multidisciplinary stone clinic in an effort to reduce her risk of future stones and preserve her remaining renal function.

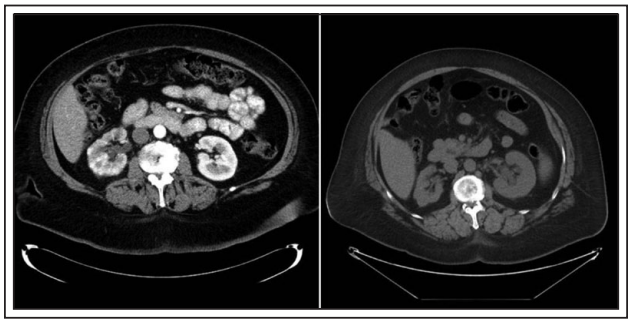

Figure 3. a) CT abdomen and pelvis with IV contrast at 3 months post-SWL showing lobulation and diffuse heterogeneous enhancement of the right kidney, **b)** CT abdomen and pelvis without contrast at 40 months post-SWL with continued lobulation and loss of renal parenchyma.

Discussion

Since its inception in 1984, SWL has remained a highly effective and relatively safe modality to treat renal and proximal ureteral stones. Although rare, devastating complications have been reported following SWL. Here we report a case of renal vein thrombosis leading to renal atrophy and functional loss in a 47-year-old, otherwise healthy but obese female. To our knowledge, this is the first report of renal vein thrombosis as a complication of SWL.

Venous thromboembolic events after SWL have been described. Brodmann and colleagues reported a case of postoperative thrombotic occlusion of the left common femoral, superficial femoral, and deep femoral veins following SWL for a left distal ureteral calculus adjacent to the area of injury. The patient was subsequently confirmed to have a heterozygous Factor V Leiden mutation.[6] Similarly, portal venous thrombosis was described following SWL of gallbladder calculi in an obese patient with history of venous thromboembolic events.[7] Superior mesenteric venous rupture was reported after SWL for a left mid-ureteral calculus in a patient with Ehlers-Danlos syndrome.[8] Interestingly, our patient had a negative hypercoagulability evaluation.

The exact mechanism of venous thromboembolism after SWL is unclear. The Virchow triad describes thrombosis to be dependent on three factors: alteration in venous flow, endothelial injury, and hypercoagulability. It has been speculated that in SWL, blood flow may become turbulent and that endothelial injury may occur, precipitating thrombosis.[5] Matlaga and colleagues utilized a porcine model to demonstrate cavitation-mediated vascular injury outside the focal zone of the lithotripter.[9] Histological studies of renal biopsy specimens obtained post-SWL have shown significant tubular and vascular changes in the plane of shock waves.[10] The microvasculature in these specimens demonstrated significant venous dilation with endothelial damage and thrombus formation. Shear forces at higher rates of shock waves may also play a role in endothelial damage and a rate of 60 shocks per minute has been shown to be optimal for treatment. Despite advances in second and third generation lithotripters including the narrowing of the focal zone for more precise administration of shock waves, collateral damage to neighboring vasculature and viscera remains a rare but unavoidable complication. It is critical to have a high index of suspicion for such complications and to counsel the patient regarding these rare potential situations prior to surgery. □

References

1. Chaussy C, Schuller J, Schmiedt E, Brandl H, Jocham D, Liedl B. Extracorporeal shock-wave lithotripsy (ESWL) for treatment of urolithiasis. *Urology* 1984;23(5 Spec No):59-66.
2. Bandi G, Best SL, Nakada SY. Current practice patterns in the management of upper urinary tract calculi in the north central United States. *J Endourol* 2008;22(4):631-636.
3. Karlsen SJ, Smevik B, Hovig T. Acute morphological changes in canine kidneys after exposure to extracorporeal shock waves. A light and electron microscopic study. *Urol Res* 1991;19(2):105-115.
4. Koga H, Matsuoka K, Noda S, Yamashita T. Cumulative renal damage in dogs by repeated treatment with extracorporeal shock waves. *Int J Urol* 1996;3(2):134-140.
5. Tse GH, Qazi HA, Halsall AK, Nalagatla SR. Shockwave lithotripsy: arterial aneurysms and vascular complications. *J Endourol* 2011;25(3):403-411.
6. Brodmann M, Ramschak H, Schreiber F, Stark G, Pabst E, Pilger E. Venous thrombosis after extracorporeal shock-wave lithotripsy in a patient with heterozygous APC-resistance. *Thromb Haemost* 1998;80(5):861.
7. Abecassis JP, Delaitre B, Morel MP, Toulon P, Pariente D, Bonnin A. Portal vein thrombosis after extracorporeal shock wave lithotripsy. *Lancet* 1991;338(8762):316-317.
8. Van der Eecken H, Schatteman P, Carpentier P, Mottrie A, Fonteyne E. Major intra-abdominal complications following extracorporeal shockwave lithotripsy (ESWL) in a patient with Ehlers-Danlos syndrome. *Eur Urol* 2002;42(6):635-636.
9. Matlaga BR, McAteer JA, Connors BA et al. Potential for cavitation-mediated tissue damage in shockwave lithotripsy. *J Endourol* 2008;22(1):121-126.
10. Willis LR, Evan AP, Connors BA, Blomgren P, Fineberg NS, Lingeman JE. Relationship between kidney size, renal injury, and renal impairment induced by shock wave lithotripsy. *J Am Soc Nephrol* 1999;10(8):1753-1762.