# Shock Wave Lithotripsy and Renal Hemorrhage

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*Although shock wave lithotripsy is a safe and efficacious treatment for nephrolithiasis, the most common acute complication is renal hemorrhage. Shock wave–induced renal hemorrhage is a potentially devastating injury if not promptly recognized and treated appropriately. The authors report a large perirenal hematoma occurring after shock wave lithotripsy and review the causes, prevention, and treatment of shock wave–induced renal hemorrhage.*
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Shock wave lithotripsy (SWL) has been safely and effectively used to treat urinary tract calculi for more than 20 years. This noninvasive procedure uses sound waves to fragment calculi, resulting in excellent rates of stone-free outcomes depending on the size, properties, and location of the stone. In many cases, SWL has become the treatment of choice for urinary calculi because of its excellent rate of stone-free outcomes, noninvasiveness, and minimal complications.

The same forces that are directed at the urinary calculi may have deleterious effects on surrounding structures. Damage to almost every abdominal organ

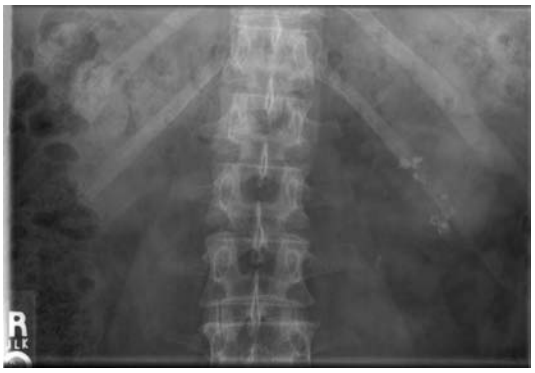


Figure 1. *Preoperative x-ray: left nephrolithiasis.*

system has been reported, but by far the most common injury is acute renal hemorrhage. The degree of injury may range from self-limited injuries to potentially life-threatening complications. Urologists must be trained to recognize these injuries and respond appropriately and expeditiously when necessary. The following case is presented to outline a potentially fatal complication of SWL and life-saving intervention; a brief review of the literature follows.

## Case Report

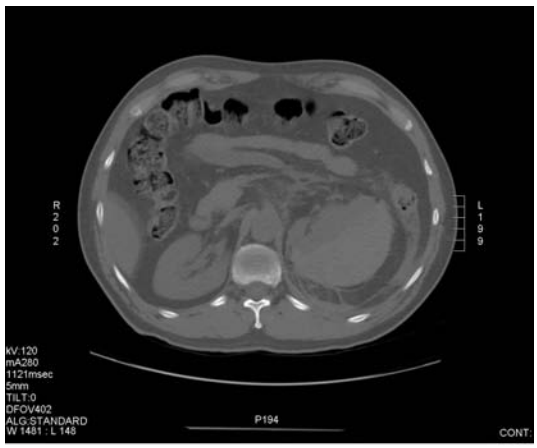
A 50-year-old man with a history of recurrent nephrolithiasis, multiple ureteroscopic stone extractions, and SWL procedures presented with mild left flank pain. He had a history of well-controlled hypertension and depression. His medications included lisinopril, paroxetine, and olanzapine. He had been smoking cigarettes (1 pack/d) for 30 years. Results from physical examination were unremarkable. Kidneys, ureters, and bladder x-ray revealed recurrent left renal stones (Figure 1).

He subsequently underwent uneventful SWL with a Siemens Dual Shockhead Lithostar lithotripter (Siemens Medical Solutions, Malvern, PA) for a 3 × 8–mm stone in the left upper ureter and 14 × 16–mm stone debris in the collecting system. One thousand shocks were delivered to the proximal ureteral stone, and 3000 shocks were delivered to the stone debris. The patient was discharged from the recovery room.

Twelve hours later, the patient presented to the emergency department with complaints of severe left flank pain and hematuria. Physical examination revealed a moderately obese Hispanic man in moderate distress. He was afebrile and tachycardic to 106 bpm, and his blood pressure was 129/96 mm Hg. Examination revealed a soft abdomen without masses and severe tenderness to palpation along the left flank with an associated ecchymosis. His laboratory values were significant for a hematocrit (Hct) (hemoglobin [Hb]) of 42.3% (14.7 g/dL), decreased from a preoperative value of 47.2% (16.3 g/dL). Prothrombin time (9.9 seconds), partial thromboplastin time (29.3 seconds), and international normalized ratio (1.1) were all within normal limits. Urinalysis revealed more than 50 red blood cells per high-power field.

The patient received intravenous (IV) fluids and hydromorphone for acute pain control. Noncontrast computed tomography (CT) scan revealed an 11 × 8–cm subcapsular hematoma (Figure 2). The patient was admitted for serial laboratory evaluations and examinations. Six hours after the initial laboratory values were obtained, Hct (Hb) had fallen to 37.4% (12.6 g/dL); 11 hours after the initial values, Hct (Hb) was 33.4% (11.9 g/dL). Because of his declining Hct and the dramatic radiographic findings, a repeat CT scan was obtained with IV contrast that demonstrated active contrast extravasation consistent with vascular injury (Figure 3). Therefore, the patient was taken urgently to the interventional radiology suite for selective embolization of the bleeding vessel. However, subsequent angiography demonstrated no evidence of active arterial extravasations (Figure 4). The patient was admitted to the hospital, and serial examinations and laboratory evaluations were continued. The patient's Hb and Hct continued to fall to a nadir of 22.0% (12.3 g/dL) on postoperative day 2. The patient received 2 U of packed red blood cells for symptomatic anemia. By the 4th hospital day his Hct had stabilized,

Figure 2. *Noncontrast computed tomography: large left perinephric hematoma.*



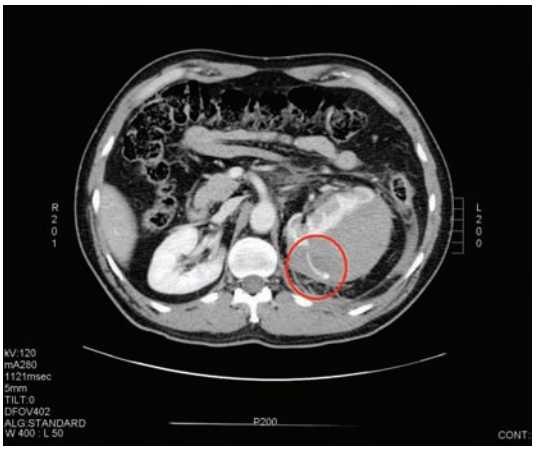



**Figure 3.** *Computed tomography scan with contrast: circle around contrast extravasation indicating active vascular injury.*

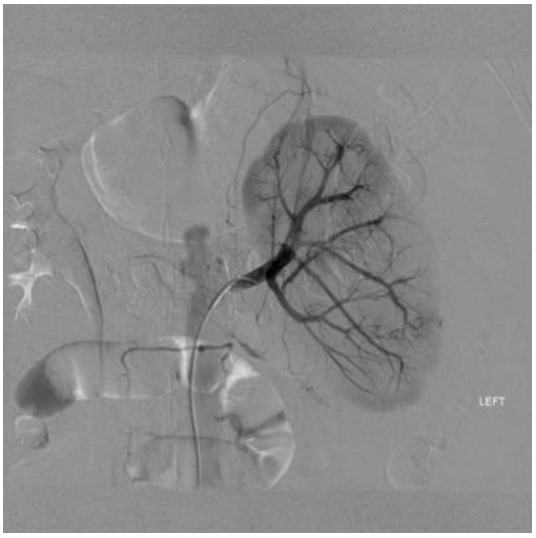



**Figure 4.** *Renal angiogram with no evidence of active extravasation.*

and he was discharged on hospital day 7.

## Review

### *Incidence*

Acute renal injury after SWL is the most common complication from this procedure, although its true incidence is unclear and poorly defined. Reports of post-SWL perirenal hematoma range from less than 1% to greater than 30%.[1-3] Much of this discrepancy is a result of differing reporting methodologies. Some investigators have only imaged patients who present with acute pain, whereas others have imaged all patients who have undergone SWL. When CT imaging has been performed routinely, hematoma formation is as high as 30%. The significance of these hematomas is unclear because only approximately 1% of patients with post-SWL hematomas are symptomatic.[4]

### *Etiology*

Although perinephric hematomas are the most common injury, other potentially devastating injuries must be considered in the post-SWL patient. The reported incidence of all extrarenal injuries combined is less than 1%.[5] Devastating injuries, including acute pancreatitis, splenic rupture, bowel injury with perforation, myocardial infarction, and rupture of abdominal aortic aneurysms, have been reported.[6-9] Additionally, less extreme injuries, such as hematochezia, scrotal ecchymosis, and injury to skeletal muscle, liver, and lung parenchyma, have also been reported after SWL.[10-13]

SWL results in acute damage to the kidney as a consequence of injury to renal vasculature as well as direct damage to renal parenchyma. The acute vascular injury includes dilation of veins, endothelial damage, thrombus formation, and disruption of renal corpuscles, which result in intraparenchymal hemorrhage and edema and may result in the formation of an extracapsular hematoma.[14] Additionally, SWL has been demonstrated to have a direct effect on the nephron: the shock waves rupture basement membranes and cells, leading to complete cellular destruction, cellular fragmentation, swelling, and necrosis, as well as the formation of intraluminal casts. The most severely damaged areas are located at F2, and the severity of damage decreases as distance from the focal zone increases.[15] Animal studies have shown SWL to result in damage and rupture to veins, small arteries, and glomerular and peritubular capillaries.[16,17] Thus renal parenchyma damage may be due to both a direct effect of the SWL and ischemia induced by local hemorrhage and disruption of the blood supply.

### *Risk Factors*

Several factors have been linked to greater rates of hematoma formation after SWL, most notably preexisting hypertension, particularly when poorly controlled. Knapp and colleagues[1] reported that hematoma formation was increased from 0.66% to 2.5% in hypertensive patients and

even further to 3.8% in poorly controlled hypertensive patients. Interestingly, multivariate analysis using mean arterial blood pressure at induction did not find this relationship to hold true.[18] In addition to hypertension, diabetes mellitus, coronary artery disease, generalized atherosclerosis, and obesity have been associated with increased rates of hematoma formation in small retrospective series.[4] The mechanism of hematoma formation has not been fully elucidated, but these findings have led some investigators to hypothesize that a loss of vascular tensile strength may be a causative factor in hematoma formation.[4]

Dhar and associates,[18] in a multivariate retrospective analysis, noted increased hematoma formation with increasing age. For every 10-year increase in treatment age, the authors note, the probability of hematoma increases 1.67 times.[18] These investigators hypothesized that changes that occur in the microvasculature with age make this patient population more susceptible to this complication.

Stone location may be related to hematoma formation as well. In one study, treatment for calyceal calculi was twice as likely to result in hematoma formation as treatment for renal pelvic calculi. This was thought to be due to the shock wave traveling through more renal parenchyma to reach the calyceal calculi than is the case with renal pelvis calculi.[18]

Additional risk factors have been noted to include underlying coagulopathy, or thrombocytopenia. Various medications that influence the coagulation cascade have been associated with post-SWL hematoma formation. Clopidogrel and aspirin, even when discontinued up to 2 weeks before treatment, have been linked to hematoma formation.[4,19-21] Although patients with bleeding diathesis are at greater risk for hematoma, this is not an absolute contraindication to SWL. Investigators have performed SWL successfully in patients with both clotting and bleeding disorders, including hemophilia, liver disease, and von Willebrand disease, by reversing these disorders before treatment.[22,23] Proper pre-SWL reversal of coagulopathy allows for safe application of this technology even in these high-risk patients.

Mechanical factors have also been linked to greater rates of hematoma formation. The type of lithotripter and number of shocks may result in a greater rate of hematoma formation, as has been demonstrated in ex vivo models.[16,17] Electromagnetic lithotripters have been reported to have marginally higher rates of hematoma formation than electrohydraulic machines.[24] Newer-generation lithotripters have small focal areas and high peak pressures and have been reported to result in higher rates of hematoma formation. The newer-generation machines have been reported by some investigators to have clinically significant hematoma rates of 3% to 12%, which is greater than the classically reported rate of less than 1% for the first-generation machines.[18,25] However, this may be due to increased imaging and more modern imaging modalities.

*Clopidogrel and aspirin, even when discontinued up to 2 weeks before treatment, have been linked to hematoma formation.*

Increasing number of shocks, shock wave voltage, and frequency of shocks have been demonstrated to result in increased renal injury and hematoma formation.[16,26-28] Animal studies have demonstrated that more than 1500 shocks resulted in an increased rate of renal injury and hematoma formation. Interestingly, pretreatment of a kidney with low-energy shock waves has been demonstrated to be protective against the renal injury and bleeding that are ordinarily caused by a typical dose of shock waves used in clinical lithotripsy.[26] Similarly, animal studies have demonstrated that decreasing the rate of shock wave delivery decreases the incidence of perinephric hematoma formation and improves stone breakage.[28]

*Presentation*

Gross hematuria invariably follows SWL, and failure of this to occur indicates a problem with shock wave delivery. Hematuria is thought to result from damage to the renal parenchyma and vasculature. It is usually self-limited and generally resolves within 12 hours, regardless of the type of lithotripter used. Persistence or worsening of hematuria may be a sign of significant renal injury and warrants further investigation.

Additionally, although flank pain is common after SWL, worsening or uncontrollable pain may be a sign of significant injury. Patients presenting with these signs require laboratory investigation including a coagulation panel, Hb, and Hct, and vital signs should be monitored. Appropriate radiographic investigation can be performed with ultrasound, CT, or magnetic resonance imaging (MRI), but CT has been demonstrated to be more sensitive in the detection of perinephric hematomas than ultrasound and is more readily available and faster than MRI; it is thus our preferred imaging modality.[2]

*Treatment*

In most clinical scenarios symptomatic perinephric hematomas are

treated with supportive care and observation. It is believed that only a fraction of those caused are ever detected, and very few data exist on the potential long-term results. Krishnamurthi and Streem[29] were not able to detect any long-term consequences in blood pressure or renal function after following a cohort of patients with known hematomas for 2 years. Furthermore, they demonstrated that the vast majority of these hematomas had resolved or were resolving in this same time frame. Further studies will need to be performed to further elucidate the potential impact that these hematomas may have on renal function and blood pressure.

Treatment should be individually tailored. The severity of subcapsular hematomas ranges from a mild contusion of the renal parenchyma to significant hematoma necessitating blood transfusion or urgent intervention, including renal embolization or even nephrectomy. The majority of these cases can be managed conservatively with admission for pain control, vital sign monitoring, bed rest, and serial determination of Hb and Hct. Coagulation studies must be obtained, and any irregularities should be corrected.

To our knowledge this is the only case in the literature with radiographic evidence of active arterial extravasation. In this case with active extravasations, on the basis of radiographic imaging and a falling Hct, a decision was made to perform angiography with the intent of performing superselective arterial embolization. When angiography was performed no further extravasation was noted, and no embolization was needed. Presumably the bleeding had been tamponaded by the renal capsule. Intervention is certainly warranted in patients who are unstable or have a rapidly declining Hct. Active hemorrhage should be recognized because it often indicates a need for urgent intervention to prevent exsanguinations. This case demonstrates, however, that active vascular extravasation may not always require intervention and may be able to be managed conservatively.

*Active vascular extravasation may not always require intervention and may be able to be managed conservatively.*

### *Prevention*

Of the many risk factors associated with post-SWL renal hemorrhage, few can be obviated, but several may be able to be mitigated. Preoperative control of diabetes and hypertension may decrease any role these medical conditions have in the formation of hematoma. Routinely we recommend continuation of antihypertensive and diabetic medication and close screening of blood pressure and blood sugars both pre- and postoperatively. We recommend discontinuation of all nonsteroidal anti-inflammatory medication at least 14 days before intervention. We routinely obtain preoperative Hct levels, and coagulation studies help to identify unrecognized risks.

## Summary

Shock wave lithotripsy is a safe and effective therapy for nephrolithiasis. The most common complication of this intervention is renal hematomas, and in very rare instances these can be potentially fatal. Recognizing this complication and treating it appropriately usually leads to good outcomes with few consequences. There is some body of evidence to suggest that patient factors, the type of lithotripter used, and the way it is applied all contribute to the formation of perirenal hematomas. In the future we hope to see more literature on prevention of hematoma formation and management when it does occur. ■

### Main Points

- Renal hemorrhage is one of the most common acute complications after shock wave lithotripsy (SWL). Most often it can be managed conservatively; in rare instances, intervention may be required.
- Preoperative evaluation of any patient undergoing SWL should include a baseline hematocrit, coagulation studies, and blood pressure and glucose monitoring.
- Patients taking anticoagulants or antiplatelet medication must stop their medication before treatment. Patients with bleeding diathesis must have their coagulation study results corrected before intervention.
- Increased age and high blood pressure are associated with increased risk of post-SWL hemorrhage. Good blood pressure control may decrease the incidence of hematoma formation.
- The type of lithotripter used and the manner in which the shock waves are delivered may influence hematoma formation. Fewer shock waves, with less energy delivered at a slower rate, decrease the risk of hematoma formation.

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