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Severe Hemorrhage after Extracorporeal Shock-Wave Lithotripsy

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Extracorporeal shock-wave lithotripsy is now commonly used for the treatment of urinary calculi (1, 2). When first-generation lithotripters are used, epidural or general anesthesia is often provided to prevent pain (3). Newer lithotripters with improved shock-wave generators and better shock-wave focusing do not cause discomfort requiring anesthesia. Treatable complications of lithotripsy, other than complications associated with anesthesia alone, have been reported, including renal parenchymal disruption, subcapsular renal hematoma formation, and, rarely, cardiac dysrhythmias, pancreatitis, liver hematomas, and hemorrhage requiring blood transfusions (2, 4-7). We report two cases of severe hemorrhage soon after lithotripsy. The first patient, who was elderly and recovering from epidural anesthesia, had postoperative hypotension, tachycardia, cardiac arrest, and death. In the second patient, the case was characterized by early recognition of bleeding, successful volume resuscitation with blood transfusions, readiness for surgical reexploration, and critical-care monitoring.

Patient 1

A 76-year-old woman with a 7-year history of hypertension had an uncomplicated lumbar epidural block before lithotripsy. This procedure was followed by cystoscopy, retrograde pyelography, and transurethral placement of a double J ureteral stent. Her vital signs were stable during treatment where she received 2400 shock-waves at 22 kilovolts.

In the recovery room, the patient's systolic blood pressure rapidly dropped from 150 to 60 mm Hg, and her heart rate increased to 150 beats/min. Resuscitative measures and diagnostic tests were begun. A 12-lead electrocardiogram showed changes presumed to indicate myocardial ischemia. The hematocrit was 0.26, 0.09 lower than the pre-operative value. Real-time ultrasound examination of the left kidney, done by a nonradiologist, failed to show changes consistent with subcapsular hematoma and dissection. During 2 hours of testing, her arterial blood pressure decreased from 120/70 to 90/60 mm Hg, and her heart rate increased from 100 to 140 beats/min. A repeat hematocrit was again 0.26.

The patient suddenly lost consciousness. A blood pressure was not obtainable and cardiopulmonary resuscitation was begun. At the start of resuscitation, the hematocrit of an arterial blood sample was 0.12. Resuscitation was stopped after normal electrolyte and arterial blood gases were obtained, but neither inotropic agents nor transvenous cardiac pacing could sustain arterial blood pressure and heart rate.

The cause of death was determined at autopsy to be massive retroperitoneal bleeding that originated from a wedged-shaped hemorrhage in the left renal cortex. The medical examiner estimated that approximately 2 to 3 litres of blood had extensively infiltrated the aortic tree and tissue planes throughout the retroperitoneum and suggested that this type of hemorrhage was not easily detectable by ultrasound imaging. There was no evidence of a recent myocardial infarction.

Patient 2

A 70-year-old man with numerous left renal calculi who had a normal routine pre-operative evaluation received 2025 shock-waves at 18 to 20 kilovolts. Two hours after treatment, the patient complained of severe left-sided flank pain. The heart rate increased to 115 beats/min. A 300 mL bolus of Ringer's lactate (Travenol, Deerfield, Illinois) was begun, but the blood pressure suddenly decreased to 80/40 and the heart rate increased to 120/min. Severe bleeding was suspected and resuscitation was begun.

Laboratory test results included a hematocrit of 0.37, down from the pre-operative value of 0.43. An ultrasound examination, made in the radiology suite, showed a large perinephric fluid accumulation around

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the left kidney. An emergent, noncontrast computed tomographic (CT) scan, which was done to stage the extent of renal injury, showed that the large perinephric fluid accumulation descended from the superior pole of the left kidney to the true pelvis. The patient was transfused with 2 units of packed erythrocytes and transferred to the intensive care unit. The patient remained hemodynamically stable and no additional blood transfusions were necessary. Serial hematocrits stabilized at 0.31.

Discussion

Although severe hemorrhage was suspected soon after the first patient's arrival in the recovery room, it could not be confirmed by renal ultrasound examinations. Consequently, volume resuscitation was delayed while other possible diagnoses were considered. Had hemorrhage been diagnosed immediately, the patient, who had a ruptured renal capsule, could have had prompt surgery for a salvage nephrectomy. This case demonstrates the importance of detecting hypovolemia before or during sophisticated imaging procedures. Although perirenal hematomas are known to occur rarely after lithotripsy (7), catastrophic renal hemorrhage is truly exceptional.

In Patient 2, precipitous hemorrhage was suspected, and resuscitative measures were begun immediately. The injury was appropriately staged with CT imaging. It is tempting to suggest that there were two risk factors for internal bleeding in these cases: the early onset and persistence of severe hematuria, and the large number of high-voltage shock-waves that were administered. Hematuria during lithotripsy is common rather than exceptional, however, and cannot be considered a signal of impending life-threatening hemorrhage. Furthermore, the voltage and number of shock-waves delivered was routine. At our institution, stone fragmentation usually occurs at 20 kilovolts or less, and the number of shock-waves applied ranges from 600 to 3000, with large stone burdens requiring values near the upper limit. These values are similar to those reported previously (2, 8).

Because life-threatening complications can develop after lithotripsy, however, thorough clinical examinations after lithotripsy are important. Although lithotripsy began as an inpatient procedure that required subsequent hospitalization, it is now usually an outpatient procedure for ambulatory patients who return home the same day. Complications that occur after discharge will be detected early if patient education and follow-up telephone contact by urology personnel provide adequate vigilance. It is also possible that some patients will visit emergency departments, primary-care facilities, and specialty-care centers soon after lithotripsy. Thus, all health care personnel should learn about the immediate and delayed complications of lithotripsy. Mild and moderate flank pain is common and is routinely tolerated with the aid of analgesics. Flank pain unresponsive to analgesics should arouse suspicion of urinary obstruction from fragmented stones, blood clots, or extraureteral compression caused by bleeding. If urinary obstruction, infection, or hemorrhage, or a combination are not treated, they can progress to pyonephrosis and septic shock, or even hemorrhagic shock, as in the cases presented.

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