

CASE REPORT

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Fatal Renal Hemorrhage After Extracorporeal Shock Wave Lithotripsy

ABSTRACT: Renal hematoma is one of the most severe complications of extracorporeal shock wave lithotripsy (ESWL). ESWL is used in the noninvasive treatment of urinary calculosis. The shock waves can lead to capillary damage, renal parenchymal or subcapsular hemorrhage. We present a case with fatal complication and the result of the medico-legal evaluation. A 71-year-old woman was treated by ESWL, renal hematoma was detected during the clinical observation, and the patient died. The death occurred despite close clinical observation as a consequence of a rare complication of ESWL. The mechanism of death was hemorrhagic hypovolemic shock due to renal hemorrhage complications due to ESWL for treatment of renal calculi. This therapeutic complication is a known complication of appropriate treatment.

KEYWORDS: forensic science, renal hematoma, extracorporeal shock wave lithotripsy, fatal outcome

Renal hematoma is one of the most significant adverse effects of extracorporeal shock wave lithotripsy (ESWL) (1,2). These study results suggest that extensive damage to blood vessels in the path of the shock waves may occur, and the primary injury can be detected in vascular tissue. ESWL is used in the noninvasive treatment of urinary calculosis. Within a few short years, ESWL revolutionized treatment of calculosis (3,4). Lithotripsy attempts to break up the stone with minimal collateral damage by using an externally applied, focused, high-intensity acoustic pulse. The successive shock wave pressure pulses result in direct shearing forces, as well as cavitation bubbles surrounding the stone, which fragment the stones into smaller pieces that then can pass easily through the ureters or the cystic duct. ESWL works best with stones between 4 mm and 2 cm in diameter that are still located in the kidney.

The shock waves themselves, as well as cavitation bubbles formed by the agitation of the urine medium, can lead to capillary damage, renal parenchymal or subcapsular hemorrhage (2). This can lead to long-term consequences such as renal failure and hypertension. The acute vascular lesion includes areas of intraparenchymal hemorrhage and edema, and could extend from the medulla to the cortex, where it forms a subcapsular hematoma. ESWL is a noninvasive treatment associated with a low complication rate; however, renal hematomas may cause severe and rarely fatal complications. ESWL is the treatment modality of choice for the majority of patients with renal or proximal urethral calculi because it is noninvasive and associated with a low complication rate; however, the renal hematomas may cause very severe or fatal complications with the necessity of careful clinical observation, assessment of quality insurance in technical processes, and medico-legal evaluation (3).

In Hungary all death cases which may be related to medical malpractice are reported to the authorities, and medico-legal

autopsy has to be performed by forensic pathologists. We present a death due to ESWL with the forensic autopsy results.

Case Report

A 71-year-old woman was treated with diabetes mellitus, ischemic heart disease, and left renal calculus. Her first ESWL was performed 6 months earlier without any complication. Her medications included insulin, aspirin, rofecoxib, and clopidogrel. The anticoagulation therapy was stopped in the hospital 2 days before the intervention. The coagulation was controlled every day. She had no fever or gross hematuria. Her vital signs and physical exam were normal. Abdominal ultrasonography and computer tomography scan (CT) demonstrated renal calculi in the left kidney. Nine hours after ESWL was performed, she complained of nausea and the physical examination detected a painful abdomen. Laboratory studies revealed normal hemoglobin and white blood cell count. Urinalysis showed 100–150 red blood cells. Due to continued pain, a CT scan of the abdomen was performed. It revealed a large (8 × 4 cm) subcapsular hematoma of the left kidney. The patient was admitted to the intensive care unit for serial hemoglobin testing and further pain management. Surgical consultations decided on conservative treatment and strict observation. Suddenly cold sweat was detected and the patient died 11 h after ESWL.

A forensic autopsy was performed 3 days after death by a forensic pathologist. The right kidney was normal. The ESWL-treated kidney had a subcapsular hematoma of the posterior renal surface. The left renal capsule was disrupted with retroperitoneal hemorrhage. The subcapsular space was filled with 925 mg of blood (Fig. 1). In the cortex and medulla, intraparenchymal hemorrhages were observed. The hemorrhages ranged in size from petechiae to large wedge-shaped areas that extended to the renal capsule, and occasionally connected to a subcapsular hematoma. Calculi fragments were found in the lower calices and renal pelvis. There was no injury in the pelvic and iliac ureter. There was no macroscopic sign of any liver disease.

Routine microscopic examination showed the characteristic features of hypertension including glomerulosclerosis, interstitial fibrosis and homogenous eosinophilic thickening of the small

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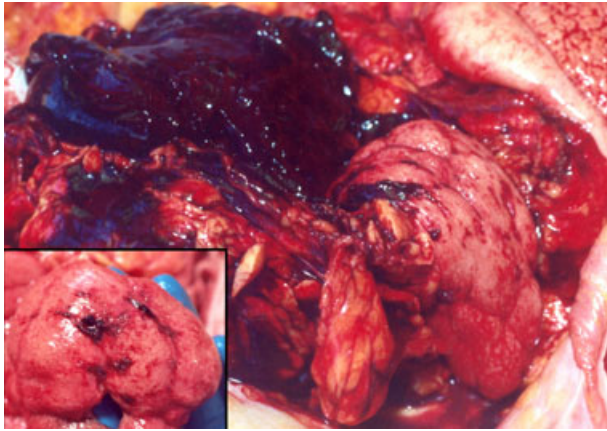


FIG. 1—Rupture of renal capsule on the left hand side, retroperitoneal bleeding and intraparenchymal hemorrhage of kidney.

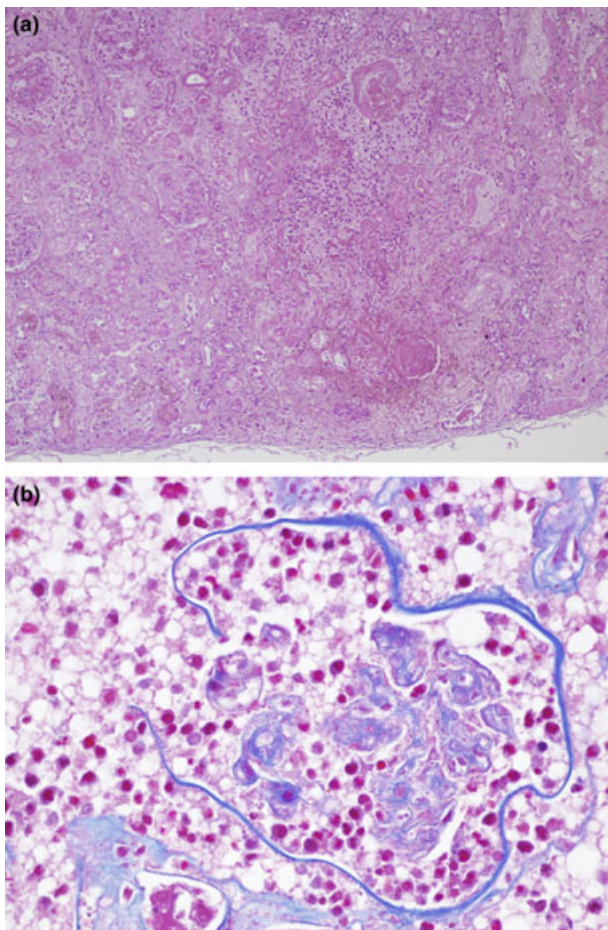


FIG. 2—(a) Interstitial and tubular hemorrhage in the subcapsular area (HE stain). (b) The Bowman's capsule is ruptured, the glomerular space is filled with cellular elements of blood (Masson trichrome stain).

artery walls. Focal hemorrhages were found in the interstitium especially in the subcapsular area (Fig. 2A). Red blood cells were found inside the tubules and glomeruli. Nearby the glomerular and tubular basement membranes were disrupted. Cellular elements of

blood filled up the glomerular space through the disrupted Bowman's capsule (Fig. 2B). The same morphological alteration was seen in the proximal tubules.

Discussion

ESWL is associated with low reported complications and mortality rates; however, renal hematoma represents one of the most severe complications (1,2). Renal hemorrhages suggest ESWL may cause renal cellular injury and ultrastructural damage in kidney tissue (2,3). In our case, the subcapsular hematoma caused death in spite of the close clinical observation. The cause of death was directly related to the renal hematoma after ESWL.

An important mechanism of ultrasound-induced nonthermal bioeffects is inertial cavitation, in which the insulated micrometer-sized bubbles expand from the negative pressure phase of the ultrasound field and then collapse in a violent implosion. Inertial cavitation plays an important role in the pathophysiology of renal injuries incurred during ESWL; however, it is unclear how tissue damage is initiated (4). Shear stresses produced by shock waves may cause the initial injury of tubules and disruption of blood vessels. The resultant bleeding from these vessels creates localized pools of blood in which cavitation activity can intensify with apoptotic effect on renal tubular cells (3,5). ESWL may cause acute vascular lesions, nephron and tubular injuries, complete cellular destruction, cellular swelling and focal necrosis, or kidney rupture. A study (3) of light and transmission electron micrographs suggests that shock waves may cause damages to the basement membranes and cells. Clinical studies demonstrated that the rate of renal hematoma after ESWL is between 0.28% and 4.1% (1,2,6–10). The incidence of renal hematoma is in Table 1.

There are several factors that predispose patients to renal rupture, subcapsular hematoma, or retroperitoneal bleeding. The most prevalent risk factor is hypertension. Atherosclerosis of the renal vasculature caused by hypertension is associated with loss of tensile strength of the vascular walls. This makes the vessel walls more vulnerable to the trauma of high-energy shock waves (6,8). Other risk factors associated with post-ESWL bleeding complications include coagulopathies, use of anticoagulant medications, diabetes, old age, coronary artery disease, and obesity.

Although gross hematuria is frequently observed after ESWL treatment and usually lasts less than 12 h, significant flank pain should alert the physician to a potential bleeding complication. The management of a patient with renal hematoma is generally conservative with pain control and measurement of urine output, hemoglobin, and creatinine levels. Therapies include transfusion of red blood cells, selective transarterial embolization (TAE) of the renal artery, and nephrectomy (1,2).

Our patient had several risk factors that predisposed her to a relatively higher rate of developing a renal hematoma after ESWL. She

TABLE 1—The frequency of renal hematoma after ESWL in clinical studies.

Source	Renal		
	ESWL (No.)	Hematoma (No.)	Rate (%)
Newman (1991) (6)	1012	6	0.59
Kostakopoulos (1995) (8)	4247	23	0.54
Dominguez Molinero (1997) (9)	Unknown	9	0.6
Collado Serra (1999) (7)	10953	31	0.28
Galleco Sanchez (2000) (1)	686	7	1.02
Riedler (2003) (2)	116	1	0.9
Dhar (2004) (10)	415	17	4.1

ESWL, extracorporeal shock wave lithotripsy.

was an elderly, insulin-dependent diabetic with coronary artery disease. The patient was managed conservatively with serial hemoglobin and kidney function studies, as well as close monitoring; however, in her case the conservative treatment was not successful. The decision on conservative and surgical therapy requires a complex risk assessment in instances of renal hematomas. The death occurred despite close clinical observation as a consequence of a rare complication of ESWL. The mechanism of death was hemorrhagic hypovolemic shock due to renal hemorrhage complications due to ESWL for treatment of renal calculi. Her diabetes, hypertension, and atherosclerosis were contributory conditions. This therapeutic complication is a known complication of appropriate treatment.

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