Thoracic spinal cord epidural hematoma after extracorporeal shock wave lithotripsy

Case report

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Extracorporeal shock wave lithotripsy (ESWL) is a well-established, safe, and effective therapeutic alternative to surgical treatment for urolithiasis. Complications of ESWL do occur in a small number of patients, and when they do, they typically involve the kidney. Formation of a spinal epidural hematoma is an extremely rare complication after ESWL for kidney stone removal. The authors present the case of a 61-year-old man in whom a large spinal epidural hematoma developed after ESWL. They discuss the possible pathogenic mechanism of such a complication. (DOI: 10.3171/2011.10.SPINE11236)

Key Words • extracorporeal shock wave lithotripsy • spinal epidural hematoma • complication

EXTRACORPOREAL shock wave lithotripsy has been the treatment modality of choice for the majority of patients with renal or proximal urethral calculi since it was first introduced in 1980. Lithotripsy attempts to break up the stone and cause 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 can pass easily through the ureters or the cystic duct. The modality works best with between 4- and 20-mm-diameter stones that are still located in the kidney.

Although ESWL is generally considered safe and effective, complications have been reported in 3% to 7% of treated patients. The major complications, including kidney rupture, psoas abscess formation, subcapsular hematoma of the spleen, acute pancreatitis, perirenal hematoma, urosepsis, venous thrombosis, biliary obstruction, bowel perforation, lung injury, aortic aneurysm rupture, and intracranial hemorrhage, have been reported to occur in less than 1% of patients.

An SEH is an uncommon clinical entity. It is defined as an accumulation of blood in the epidural space that can mechanically compress the spinal cord. The overall incidence of SEH is very low. Many causes of SEH have been described, including coagulopathy, trauma, surgery, and vascular lesions.

To our knowledge, we present the first case of thoracic spinal cord epidural hematoma occurring after ESWL for a right-side urinary stone; the hematoma was treated by surgical decompression.

Case Report

This 61-year-old man presented to our emergency department with bilateral lower-extremity weakness and voiding difficulty. Early the same day, he had undergone ESWL for a right-side ureteral stone. He had a 5-year history of hypertension for which he received antihypertensive medication and was not taking anticoagulants or herbal medicine. He began to feel severe midback pain and pain radiating to both legs 6 hours after ESWL. By 10 hours, he could not walk. There was no evidence of coagulopathy on inspection of laboratory findings. On neurological examination, he had Grade 3 weakness in both legs, hypesthesia below the T-10 level, pathological reflexes in both legs, and a decreased anal tone. Thoracic CT scanning demonstrated a hematoma causing cord compression. Thoracic MR imaging revealed a T10–L1 epidural hematoma (Fig. 1). We performed an emergency
total laminectomy of T-10, T-11, and T-12 and removed the hematoma. On follow-up sagittal and axial MR imaging, we noted no cord compression (Fig. 2). The patient’s gait disturbance progressively improved after surgery. Twelve months postoperatively, the patient was neurologically improved, and he was able to walk without the aid of a cane.

Discussion

Since it was first introduced in 1980, ESWL has been the treatment modality of choice for the majority of patients with renal or proximal urethral calculi. Although it is generally considered safe and effective, complications have been reported in 3% to 7% of the patients. Posttreatment complications of ESWL include transient gross hematuria, flank pain, urinary tract infection, and hematoma, which usually can be treated conservatively. An extremely small number of cases, however, require transfusion or surgical treatment after clinically significant hemorrhage has developed. The major complications, including kidney rupture, psoas abscess formation, subcapsular hematoma of the spleen, acute pancreatitis, perirenal hematoma, urosepsis, venous thrombosis, biliary obstruction, bowel perforation, lung injury, aortic aneurysm rupture, and intracranial hemorrhage, have been reported to occur in less than 1% of patients.

The mechanism of hematoma formation is related to the piercing effect of a liquid microjet, which has an impact velocity of more than 200 m/second and occurs as the tiny bubbles and the shock wave interact. Inertial cavitation is an important mechanism of ultrasound-induced nonthermal bioeffects. It plays an important role in the pathophysiology of renal injuries incurred during ESWL; however, it is unclear how tissue damage is initiated. Shear stresses produced by shock waves may have a direct effect on the renal vasculature and cause direct damage to renal parenchyma. Acute vascular injury results in intraparenchymal hemorrhage and edema and may cause formation of an extracapsular hematoma. Once the hemorrhage occurs, the weight of the blood clot cuts off the vessel between the renal parenchyma and the capsule. Thus, the hematoma increases and localizes pools of blood in which cavitation activity can intensify with an apoptotic effect on renal tubular cells.

Several factors have been linked to greater rates of renal rupture, subcapsular hematoma, retroperitoneal bleeding, or bleeding in other locations after ESWL. Hypertension, generalized arteriosclerosis, clotting disorders, a previous ESWL, advanced age (> 60 years), diabetes mellitus, coronary artery diseases, and obesity have all been proposed as significant risk factors. The most prevalent risk factor is hypertension when poorly controlled. The mechanism of hematoma formation has not been fully elucidated, but some investigators have hypothesized that these factors are associated with loss of tensile strength of the vascular walls, and a loss of vascular tensile strength may be a causative factor in hematoma formation. Our patient had several risk factors such as hypertension and misdirection of the ultrasound field to the spinal cord that predisposed him to epidural vessel rupture, which led to a spinal epidural hematoma after ESWL.

Spinal epidural hematoma is an uncommon clinical entity. It is defined as an accumulation of blood in the epidural space that can mechanically compress the spinal cord. Compressive SEH is acute and progressive. Improperly managed, it can cause permanent neurological deficits. The overall incidence of SEH is low. The incidence of SEH as a complication of epidural anesthesia is estimated to be between 1 in 150,000 and 1 in 190,000. Spontaneous SEH accounts for less than 1% of all spinal space-occupying lesions.

Three mechanisms of SEH formation are discussed in the literature: rupture of epidural veins, rupture of epidural arteries, and hemorrhage resulting from vascular anomalies. Clinical symptoms of SEHs usually present with acute, severe pain in the location of the hemorrhage with pain radiating to the extremities. Within hours, motor and sensory deficits are observed, with paraplegia or paralysis (usually flaccid paralysis with hyporeflexia) and urinary retention. Lepoire and coworkers reported a mean time of 3 hours between onset of pain at the location of hemorrhage and full clinical presentation. However, in some cases, full clinical presentation was observed after 18–20 hours or even 2–3 days. In our case, clinical symptoms developed 12 hours after ESWL.

Clinical evaluation of pain control and neurological dysfunction is of utmost importance in early diagnosis. Currently, MR imaging is the diagnostic method
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of choice, although CT myelography remains an option.\(^\text{1,12,15,19}\) Magnetic resonance imaging is noninvasive and allows better differentiation among tumor, infection, disc herniation, and hematoma. It is also possible to demonstrate the complete extent of the lesion.

The most consistently reported management strategy for symptomatic SEH in medically stable patients is surgical exploration and decompression of compressive lesions. Decompressive laminectomy is the most common technique of SEH evacuation in all parts of the spine, and it is the most recommended procedure in many publications for surgical treatment of spinal canal hematomas.\(^\text{5,9,12}\)

**Conclusions**

The authors documented a very rare complication of ESWL. Urologists should pay attention when performing ESWL in patients with risk factors such as hypertension, coagulopathies, anticoagulant medication use, diabetes, old age, and coronary artery disease. Urologists and neurosurgeons alike should be aware of this rare complication, because prompt diagnosis and essential treatment can prevent significant neurological deficits.

**Disclosure**

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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