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Reversible acute renal failure after unilateral extracorporeal shock-wave lithotripsy

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Abstract Extracorporeal shock-wave lithotripsy (ESWL) is the treatment of choice for the majority of patients with renal or proximal ureteral stones. We describe an unusual case of anuric renal failure following ESWL, in absence of obstruction or myoglobinuria, in the presence of unilateral nephrolithiasis and two normally functioning kidneys. A mechanism for this patient's acute renal failure (ARF) is postulated. Although the frequency of ARF after ESWL is extremely rare and the mechanism responsible for ARF is not understood, the appearance of ARF, when ureteral obstruction or hematoma are absent, should be included among complications following EWSL. Attention should be paid to older patients.

Keywords Lithotripsy · Nephrolithiasis · Acute renal failure · Treatment complications

Introduction

Unilateral extracorporeal shock-wave lithotripsy (ESWL) is the treatment of choice for the majority of patients with renal or proximal ureteral stones. Although many patients who undergo ESWL experience transient hematuria, the incidence of major complications is low [1].

We describe an unusual case of anuric renal failure following ESWL, in absence of obstruction or myoglobinuria, in the presence of unilateral nephrolithiasis and two normally functioning kidneys. A mechanism for this patient's anuric renal failure is postulated.

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Case presentation

A 68-year-old man with a history of right nephrolithiasis was admitted for right flank pain. Renal function was normal (Cr: 1.2 mg/dl). Abdominal ultrasound revealed two kidneys of normal size with preserved cortex and the presence of a right pyelic stone 1.5 cm in diameter. Intravenous urogram confirmed the diagnosis; no significant obstruction was present. ESWL (Sonolith 3000) was then performed under monitored anesthesia care because of the patient's pain intolerance, using boluses of propofol (0.5–1 mg/Kg). Ketoralac 30 mg and meperidine 20 mg were administered for pain control. A total of 3400 shock waves (15.5 Kv) was administrated. In the immediate postoperative period the patient experienced nausea and gross hematuria. He was treated with ketoralac 30 mg for analgesia. Two days later he developed oligoanuria. Blood chemistry showed that serum creatinine had risen to 9.1 mg/dl. Physical examination, blood pressure and body temperature were normal. He had no significant risk factors (hypertension, diabetes mellitus, arteriosclerosis, coronary artery disease, obesity). Serum creatinine phosphokinase was in the normal range and no anemia, low platelet count or haemolysis were noted.

Color Doppler ultrasound confirmed the disintegration of the stone and revealed an increase of corticomedullary demarcation and hyperecogenic parenchyma. No intrarenal, subcapsular or perirenal hematoma were observed. Ureterohydronephrosis was not present. Renal perfusion was normal, but resistive indexes were increased ($RI > 0.75$) bilaterally (Fig. 1).

The patient was treated conservatively with saline, serum bicarbonate and furosemide. Urine analysis was negative. Renal biopsy was not performed because renal function recovered within 4 weeks. Diagnosis of acute tubular necrosis (ATN) was made.

After a 6-months follow-up the renal function is slightly decreased (serum creatinine: 1.5 mg/dl) and the blood pressure is normal. A repeated color Doppler ultrasound demonstrated normal kidneys with resistive index in the normal range (Fig. 2).

Discussion

Major complications after ESWL are rare and related to the administration of shock waves or obstruction by stone fragments. Clinically significant perirenal or subcapsular hematoma is present in less than 1%, urinary tract obstruction and urinary infections occur in 10% of cases [2].

Although recent reports on animals indicate that a dose of shock waves sufficient to comminute a stone will

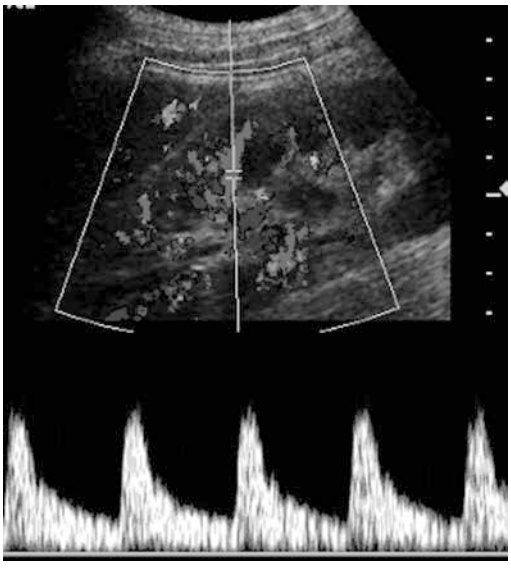

Fig. 1 Color Doppler ultrasound (HDI 5000 ATL, USA equipped with a 3.5-MHz convex probe) reveals a normal renal perfusion with increased resistive indexes (RI > 0.75)

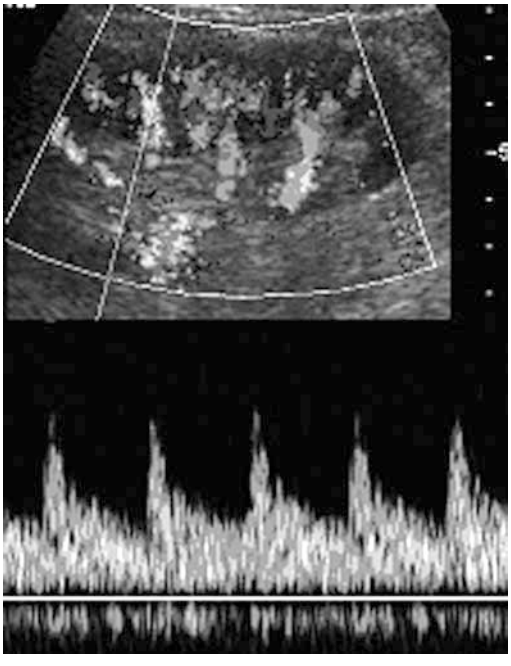

Fig. 2 Six months after the procedure a repeated color Doppler ultrasound demonstrates normal kidneys with resistive index in the normal range

always damage the kidney, the lesion produced is largely limited to the region occupied by the zone of peak pressure [3]. The morphological lesions included dramatic vascular insult. Tubular and interstitial cell injury occurs in the region of the vascular lesions. Parenchymal damage appeared correlated to the dose of shock wave insult.

In a controlled canine model, a vacuolization of tubular cell, which may signify the start of tubular necrosis, was a constant finding even in the contralateral, non-exposed kidney [4].

Experimental works demonstrate that ESWL may cause a reduction in renal plasma flow, both in the treated and contralateral kidney. In non-exposed kidney, however, the reduction of renal flow is only about one third as much as occurs in the ESWL-treated kidney and recovers within 4 h [5]. This vasoconstriction is not dose dependent with regard to the voltage or number of shocks, suggesting that the mechanism inducing this effect may be different from that causing parenchymal damage. Literature in renal ischemia suggests that loss of blood flow for even a fairly short period of time can cause tubular damage.

A group of investigators demonstrated that, in humans, the pathologically high intrarenal resistive index in treated kidney was correlated with systemic blood pressure. Age was a risk factor too [6]. Data in the literature demonstrate only a slight and transient increase in renin activity without any modification of endothelin [7] suggesting that other mechanisms, like autonomic nervous system, may play a role in described haemodinamic modifications. Studies in human subjects have shown that reflex maneuvres expected to produce changes in efferent renal sympathetic nerve activity (ERSNA) are associated with alteration in renal blood flow and vascular resistance. Moreover, reflex-increasing ERSNA determine a direct change in renal sodium and water handling with antidiuresis without alteration in glomerular filtration rate and renal blood flow. Acute experimental studies involving anesthesia and surgical trauma likely increase the basal level of ERSNA [8].

Our patient was 68 years old and he was never hypertensive either during acute renal failure (ARF) or during the follow-up. The intrarenal resistive index was pathologically elevated in both kidneys during ARF, but returned to normal at follow-up. This suggests that reduction in renal blood flow may be the cause of ARF, although a high resistive index is also noted as a consequence of interstitial edema or other causes of ATN. Hypoperfusion is therefore associated with a graded parenchymal injury ranging from none in prerenal azotemia to frank ATN and cortical necrosis. In this patient, the clinical course with recovery in few weeks showed that ATN resulted from haemodynamic instability probably aggravated by administration of non-steroidal anti-inflammatory agent. Ketoralac is a known nephrotoxic medication because it inhibits prostaglandin synthesis, thereby decreasing renal perfusion. Although the incidence is extremely small, ARF from ketoralak received during surgical insult has been reported [9]. On the other hand, the patient received only two doses of ketoralac and had a good renal function preoperatively.

Only six cases of ARF after unilateral ESWL in two normally functioning kidney are reported, but the doses of shock waves were higher and patients experienced an increase in serum creatinine phosphokinase and myoglobulin [10].

In our patient, rhabdomyolysis can not be postulated, although it is not possible to exclude that ''toxins'', like hemoglobin, released from the area of renal contusion,

can produce vasoconstriction by inhibiting the production of endothelial relaxing factors. Furthermore, ferrous iron compounds catalyze the formation of toxic free radicals. Renal damage could result from imbalance between O2 supply, limited by renal vasoconstriction, and continued O2 demand for inefficient transport. Synergism between renal hypoperfusion and toxic insult may predispose to more severe oliguric ARF.

Unexplained, however, is the extreme rarity of ARF after unilateral ESWL, while ESWL is commonplace. It is possible that, in the majority of cases, reduction of renal blood flow is so short that it is underestimated. Moreover, it is possible that a reduction in renal blood flow may occur only in patients with an abnormality in the autonomic nervous system involving exaggerated response to certain visceral stimuli.

Although the frequency of ARF after ESWL is extremely rare and the mechanism responsible for ARF is not understood, the appearance of ARF, when ureteral obstruction or hematoma are absent, should be included among complications following ESWL.

Attention should be paid to older patients, especially if older than 65. In these patients use of ketoralac should be avoided.

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