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Case report

A Neurosurgical Patient with Acute Renal Failure

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Abstract

Rhabdomyolysis is a common cause of acute renal failure. It represents a peripheral muscle damage with myoglobinuria and electrolyte disturbances with or without volume changes and renal injury. We present a 47-years-old male patient with traumatic subdural hematoma who developed acute renal failure after two grand mal seizures. After alkalization, hydration and diuretic treatment renal failure subsided and the patient underwent successful evacuation of hematoma. We discuss the pathogenic mechanisms of development of acute renal injury in rhabdomyolysis.

Keywords: acute renal failure, rhabdomyolysis, epileptic seizure, traumatic brain injury, traumatic subdural hematoma

Introduction

Rhabdomyolysis (RM) is striate muscle-cell damage with a release of intracellular constituents to the circulation, myoglobinuria, electrolyte abnormalities, with or without subsequent kidney injury [1]. The main intracellular constituents that are released in circulation after myocyte damage are myoglobin, creatin phosphokinase (creatin kinase) and lactate dehydrogenase, along with potassium and phosphates [1]. Myoglobin is a muscular cell oxygen-binding protein with molecular weight of 18.8 kDa that binds plasma proteins and small amounts reach urine. In massive striate muscle damage, plasma proteins cannot bind the excessive amount of myoglobin and the latter reaches the urine. In acidic environment, urinary myoglobin can precipitate and cause tubular obstruction and toxicity leading to an acute kidney injury (AKI) [1]. The injured muscle fibers can sequestrate large amount of fluid in the form of muscle cell oedema that leads to a decreased effective circulating volume and renal hypoperfusion that can further exacerbate the kidney injury [2]. Moreover, the efflux of fluid to the injured muscular fibers may lead to a severe hypokalaemia with an additional impact on renal tubules. The striate muscle damage can also cause severe hyperkalaemia, hypercalcemia and hyperphosphatemia with hyperuricemia that can have additional harmful effects on the renal tissue [3,4]. Another characteristic laboratory sign of rhabdomyolysis is the development of metabolic acidosis with increased anionic gap (due to release of organic acids from the disintegrated muscle cells) [3,4]. RM is one of the leading causes of AKI, associated with up to 10% of the cases every year [3]. The true prevalence of RM worldwide is unknown and is probably significantly underestimated [3], because in the majority of cases it undergoes spontaneous resolution. Approximately 26 000 cases of RM are reported in the US every year [3]. Moreover, the incidence of AKI in RM patients remains unclear. The estimated incidence of this complication is 4-33% of all RM patients [1,3].

RM can be classified in two main subtypes: traumatic (muscle trauma) and non-traumatic (associated with seizures or strenuous physical activity, muscle compression, alcohol and/or drug abuse, dehydration, and inborn metabolic defects). No matter the cause, up to one third of RM patients can develop AKI, presenting with the following clinical signs and symptoms: pain, oedema and muscle weakness of the injured sites, myoglobinuria (pinkish to reddish discoloration of the urine), and decrease in the urine volume to anuria [1-4].

We present a patient with acute renal failure due to rhabdomyolysis, associated with seizures at the background of traumatic acute subdural haemorrhage.

Case presentation

A forty-seven years old male patient was admitted to the Clinic of Neurosurgery for evacuation of traumatic fronto-parietal subdural hematoma that developed after blunt head trauma. At the admission the patient was brady-psychic but adequate contact and showed no neurological deficit. Vital signs were within the normal limits. The physical examination showed no ab-

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normalities, except for hematoma on the left side of his forehead. He reported hitting his head after he fell down in the afternoon of the day before the admission. No loss of consciousness after the trauma was reported. Approximately 6 hours after the trauma he had a grand mal seizure and was taken to another hospital where he had a second seizure and underwent computed tomography of the head that detected small subdural hematoma in the left fronto-parietal area. The patient was referred to the Clinic of Neurosurgery in our hospital for evacuation of the hematoma and on the way to Neurosurgery he developed a third grand mal seizure. The patient reported pinkish discoloration of the urine the morning at the admission.

The initial laboratory investigations revealed increased white blood cell count of 14.5 G/l, serum creatinine of 155 mcmol/l, urea of 16 mmol/l, uric acid 540 mcmol/l, potassium 6.8 mmol/l, sodium 139 mmol/l, chlorides 96 mmol/l, calcium 2.92 mmol/l, phosphates 2.6 mmol/l, AST 240 U/l, ALT 63 U/l, creatin kinase 10 008 U/l. Urinalysis revealed positive protein and blood in the urine with no red blood cells in the sediment. Alkaline base balance revealed mild metabolic acidosis (pH of 7.28, base excess of (-10), standard bicarbonate of 16 mmol/l) with increased anion gap of 33.8 mmol/l. Serological markers for viral hepatitis (HAV, HBV and HCV) were negative.

Abdominal ultrasound revealed increased kidney size with widened and slightly hyperechogenic parenchyma with marked hypoechogenic pyramids (Figure 1).

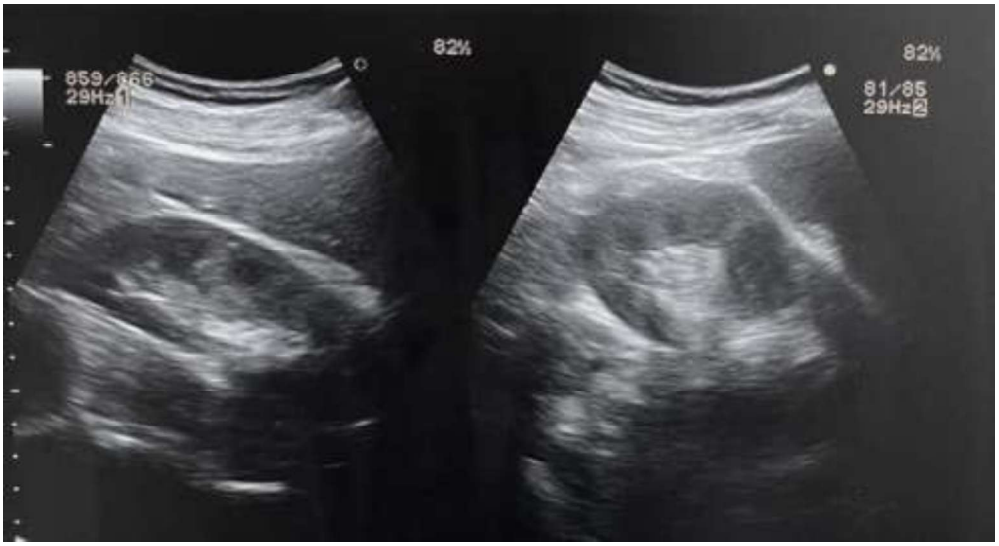

Fig. 1. Abdominal ultrasound – enlarged kidneys with thickened slightly hyperechogenic parenchyma and visible pyramids

The patient was diagnosed with RM-associated AKI, caused by symptomatic epileptic seizures due to acute subdural hematoma. He was initiated on intravenous infusions with saline and alkalization with intravenous sodium bicarbonate 60 ml, intravenous furosemide 40 mg and underwent an emergency evacuation of the hematoma. After the operation, at the background of saline infusions, intravenous alkalization and diuretic administration, renal function improved and creatinine, urea and uric acid levels returned to normal limits within 4 days. The hyperkalaemia, hypercalcemia, hyperphosphatemia and the acid-base balance also normalized. Cytolytic enzymes and urine investigations normalized within 7 days, while the renal parenchymal echogenicity returned back to normal within 14 days.

The postoperative CT revealed no residual collection, the patient was put on prophylactic anti-epileptic treatment with carbamazepine. One year after the surgery the patient remains seizure-free, with normal renal function and urinary dip-stick tests and normal cytolytic enzymes. The control CTs revealed no residual collections.

Discussion

Rhabdomyolysis is among the most common causes of AKI, especially in neurological and neurosurgical patients. The key mechanisms of renal injury in these cases include [1-4] an oxidative stress due to iron overload of the tubular cells in myoglobinuria, inflammation and apoptosis of tubular cells due to the oxidative injury, acute tubular obstruction in myoglobinuria and increased uricosuria, renal hypoperfusion due to decreased circulating volume (developing due to oedema of the necrotic muscle fibres), vasoconstriction due rennin-angiotensin-aldosterone system activetion in hypovolemic state, and dyselectrolitemia-hypokalaemia due to efflux of potassium in the necrotic muscle fibres, or hyperkalemia, hyperphosphatemia and hypercalcemia due to liberation of ion from the necrotic muscle cells.

If treated properly, renal damage in RM can be reversible. The recommended therapeutic strategies in RM include [1-3,5] general supportive measures, renal replacement therapy, anti-inflammatory medications (corticosteroids), iron chelators, antioxidants and N-acetyl cysteine, recombinant human erythropoietin and mesenchymal stem cells in particular cases of mice models, but no studies have been performed in humans.
The mortality in RM is between 2% and 46% [1,3], depending on the aetiology, timely treatment and comorbidity. The renal recovery depends on the timely treatment and the severity of RM, determining the extent of subsequent development of tubule-interstitial and glomerular fibrosis. At present, yet, no studies on the long-term outcome of RM in humans have been performed [1,3].
In our patient, we observed mild AKI with predominantly extrarenal uraemia (more marked increase in urea and uric acid levels than of creatinine levels), hyperkalemia, hypercalciemia and hyperphosphatemia, metabolic acidosis with increased anion gap (calculated according to the following formula: $[Na^+ + K^+] - [Cl^- + HCO3^-]$, normal levels 4-12 mmol/l [6]) due to the liberation of intracellular constituents in the circulation, increased cytolytic enzymes (AST>ALT, creatin kinase), false positive blood on urine dipstick test (due to the presence of myoglobin in the urine). The abdominal ultrasound scan revealed the typical renal image of rhabdomyolysis-associated renal injury-enlarged kidneys with thickened parenchymal zone and increased parenchymal echogenicity-due to precipitation of myoglobin within the tubular lumens [7]. The RM was caused by three grand mal seizures associated with head trauma with subdural hematoma. The timely diagnosis and the proper and timely treatment of both, neurosurgical and nephrological emergencies lead to fast and complete recovery. As the prevalence of post-traumatic seizures can be as high as 24% in acute and 11% in chronic [8] subdural hematomas, RM should be considered as an important cause of AKI in such patients and prophylactic anti-convulsant treatment should be initiated in all cases of acute subdural hematomas [9].
Multiple cases of seizure-associated traumatic RM with ARF have been described in the literature. Mishra et al. reported a patient with acute renal failure (ARF) after epileptic seizure [10]. L. Wang *et al*., present a case of rhabdomyolysis and hyperuricemia leading to ARF in a patient with status epilepticus [11]. In both cases the patients required acute temporary dialysis due to conservatively not manageable oligoanuria. Kara *et al*., also report a young patient with AKI following seizure-induced RM that required temporary dialysis [12]. The extracorporeal blood purification and conservative treatment (rehydration, alkalization and forced diuresis) lead to the restoration of kidney function. The first and the second case underline the role of hyperuricemia for the development of renal injury in RM and the third patients revealed the importance of an early diagnosis and timely treatment for the prognosis of ARF. Singh *et al*., also report a young patient with RM-induced ARF [13] on the day after developing two short lasting tonic-clonic seizures. Aggressive rehydration lead to a fast restoration of renal function and decrease in both cytolytic enzymes and creatinine levels without the need of extracorporeal blood purifycation. Our patient also developed ARF with hyperuricemia due to seizure-associated RM. The early diagnosis and initiation of the conservative treatment, including forced alkaline diuresis (saline infusions, alkalization and stimulation of diuresis with furosemide), lead to a rapid restoration of renal functional capacity without the need for acute dialysis.

Conclusion

In conclusion, in patients with ARF at the backgound of seizures and trauma, the timely and adequate diagnosis and treatment (including rehydration and forced alkaline diuresis) are of crucial importance for preservation of the renal function and patient's life and require the team efforts of neurologists, neurosurgeons, anaesthesiologists and nephrologist.

Conflict of interest statement. None declared.

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