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Case Report

Rhabdomyolysis and acute kidney injury in the deceased donor: a case report

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ABSTRACT

Rhabdomyolysis is a common complication among brain death donors, affecting the number of organ donations and the quality of donor kidneys. Case report: Male, 17 years old, admitted to the hospital due to a car accident. Subsequently, brown urine appeared, blood myoglobin increased significantly, urine output decreased, and renal function impaired. Treatments including fluid replacement, alkalization of urine, plasma exchange and bedside CRRT were given. The patient's renal function recovered, and the organs were successfully acquired. The renal function recovered well after transplantation. Conclusion: Attention should be paid to rhabdomyolysis. Early diagnosis and treatment of patients with brain death could improve donation success rate and the recovery of postoperative renal function.

Keywords: Acute kidney injury, Donor, Rhabdomyolysis, Transplantation

INTRODUCTION

Rhabdomyolysis (RM) refers to striated muscle cell injury caused by various causes, which releases muscle cell components such as myoglobin and creatine kinase (CK) into the blood, further causing metabolic disorders and organ dysfunction syndrome.[1] Acute kidney injury and arrhythmia are the most common complications. At present, Chinese organ donors are the only source of transplanted organs. However, the probability of rhabdomyolysis among organ donors with brain death is higher than that of other patients and should be taken seriously.[2] Rhabdomyolysis would cause renal tubular obstruction for organ donors, resulting in acute kidney injury. Many donor kidneys are thus abandoned, aggravating donor organ shortage.[3] Can such patients also serve as donor kidneys? There are very few reports on this situation in the literature. This paper reported a case of traumatic coma with rhabdomyolysis, in which the kidney was successfully transplanted to uremic patients after appropriate monitoring and treatment.

CASE REPORT

The 17-year-old male patient was injured in a car accident at about 12 am on June 08, 2017. He was hit on the head and face, accompanied by coma. There was bloody fluid in the nasal cavity, and no convulsions, fever, vomiting or cough were observed. He was sent to the local hospital emergency department by ambulance, where CT examination showed subdural hematoma in the left subdural area, brain swelling, subarachnoid

hemorrhage, multiple fractures in craniofacial bone and left zygomatic arch as well as local scalp swelling and lung contusion at both sides. The boy was enrolled in the hospital as "extraordinary intracranial injury" for cranial hematoma removal and decompressive craniectomy. The patient was transferred to ICU for further rescue and intensive care, with ventilator assisting breathing. Treatments such as ECG monitoring, hemostasis, dehydration, hormones, anti-infection and other aids were given. However, the patient was still in a deep coma with unstable vital signs. Ventilator assistance was needed to keep ventilation, and booster drugs were used to maintain blood pressure. Considering the critical condition and poor prognosis of the patient, his family decided to transfer him to ICU in our hospital for further treatment against "severe intracranial injury".

Physical examination: The patient's heart rate was 101 beats/min, blood pressure 101/64 mmHg, and blood oxygen saturation 98%. The patient was deeply comatose, and the pupils on both sides were dilated, with the light reflection disappearing directly and indirectly. Corneal reflex, ocular reflex, vestibular reflex, and cough disappeared. No spontaneous breathing, with continuous ventilator assisting breathing. The urine color was brown, as shown in Figure 1. The urine volume was small, and the urine volume per hour was 20 ml/hr.

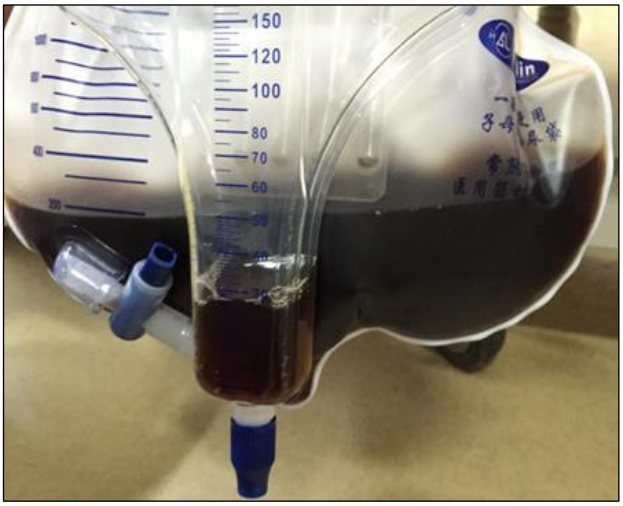

Figure 1: Brown urine from organ donors with rm.

Related auxiliary examination: blood creatinine 275umol/L, blood myoglobin 12650ug/L.

Admission diagnosis

1. i Subdural hematoma in the left frontal apex;
   ii Brain contusion;
   iii Subarachnoid hemorrhage;
   iv Cerebral palsy formation;
   vi Skull base fracture;
2. Lung contusion at both sides;
3. Lung infection;
4. Hypernatremia;
5. Hypoproteinemia;
6. Acute kidney injury;
7. Rhabdomyolysis

Treatment

After admission, the patient was given treatments including continuous ventilation, anti-infection, vasoactive drugs to maintain blood pressure, and the patient was given sodium bicarbonate to alkalize urine, plenty of fluids and appropriate use of diuretic mixture. Bedside plasma exchange was performed twice, and bedside CRRT treatment was also given.[4] After the treatment, the patient's urine volume, creatinine (Cr), urea nitrogen (BUN) changed to the level as shown in Figure 2.

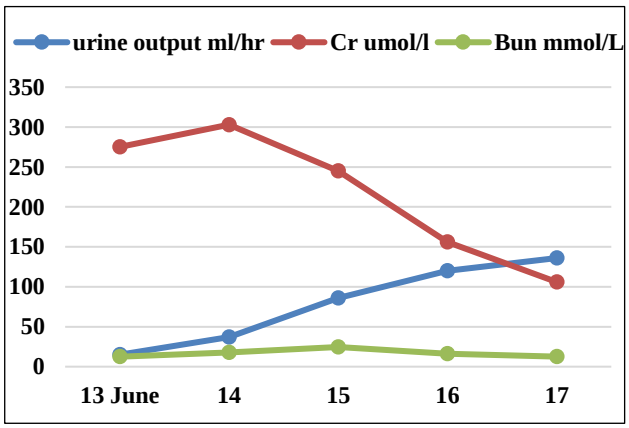

Figure 2: Renal function changes during treatment.

WBC, HB, PLT, Na+, K+, myoglobin, UMb, CK-MB are shown in Table 1. Subsequently, the patient passed the criterion for brain death, and his family agreed to donate organs.[5] Organ removal was implemented. The kidney was dark, and biopsy was performed for pathological examination, as shown in Figure 3.

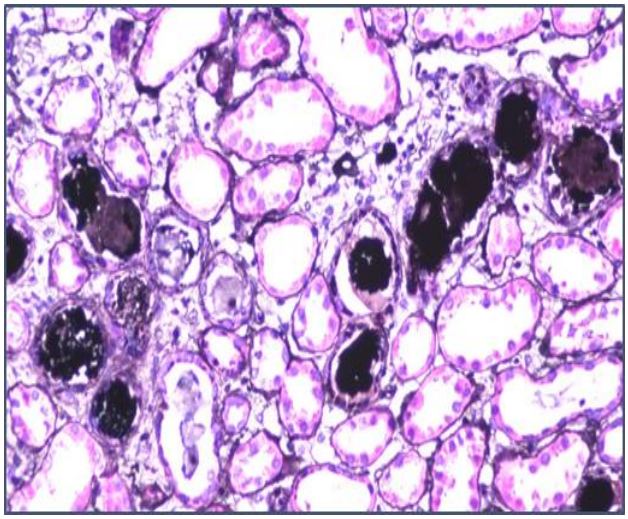

Figure 3: Pathological picture of donor kidney.

Two donor kidneys were transplanted to two uremia patients, and the renal function of the patients recovered well after transplantation without delayed recovery, as shown in Table 2. The transplant was successful.

Table 1: Indicator changes of donor.

Date	June 13	June 14	June 15	June 16	June 17
WBC (10E/L)	15.43	10.68	18.44	20.59	24.35
HB(g/L)	86	98	125	126	139
PLT (10E/L)	100	63	38	53	35
Na^+(mmol/L)	201	189	176	162	144
K^+(mmol/L)	5.08	3.24	4.86	4.27	4.63
Myoglobin(ug/L)	12650	5252	3310	1515	1719
UMb (ug/L)	1361	1554	74980	10000	70170
CK-MB (ug/L)	41.6	14.2	4.94	5.08	2.68

DISCUSSION

Rhabdomyolysis syndrome (RM) refers to a series of clinical syndromes caused by the destruction of membrane after the damage of rhabdomyolysis cells, and the release of myocyte substances into extracellular fluid and blood circulation.[6] The main manifestations include myalgia, limb weakness, tawny urine, often complicated with electrolyte disorder, acute renal failure (ARF), which poses a serious threat against people's health. According to statistics, about 10% to 40% of patients with rhabdomyolysis developed acute renal failure, in which rhabdomyolysis accounted for about 15% of the causes. Previous studies have shown that children with rhabdomyolysis have a higher incidence of acute renal failure at 42% to 50%. Approximately 26,000 cases of RM are reported in the United States each year. In recent years, the number of cases reported in China has gradually increased, drawing people's attention to RM study.

There are many causes of RM. Traumatic factors are not rare in surgery, while the incidence of internal causes is much higher than that of surgery. Non-traumatic factors, such as stroke, poisoning and infection are the common causes of RM. Some scholars divided the causes of RM into physical and non-physical categories. Physical causes include compression, trauma, sports and excessive muscle activity, high fever, etc. Local tissue ischemia caused by tourniquet, corset, embolism, hypotension, shock and osteofascial compartment syndrome may also contribute to RM. It might also come from war, earthquake, landslide, car accident, electric shock, burn, etc. Excessive exercises, such as intense training of athletes and soldiers, dystonia, epileptic, may result in RM. In addition, excessive heat, such as heat stroke and malignant hyperthermia may cause RM as well.[7]

Non-physical causes mainly include drugs, infections, poisons, electrolyte disorders, endocrine and genetic metabolic diseases and autoimmune diseases. Drugs with lipid-lowering, antipsychotic and antidepressant, sedative, hypnotic, anti-inflammatory and nucleoside antiviral effects, antihistamine and morphine may play a part in RM. Virus infection: influenza viruses A and B, cytomegalovirus, herpes zoster virus, herpes simplex virus, HIV, coxsackie virus, E-B virus, chicken pox virus, parainfluenza virus, etc. Poisons: CO poisoning, alcohol poisoning, organophosphorus pesticide poisoning, biting by venomous snake, eating poisonous mushrooms or crayfish, etc. Severe electrolyte and endocrine disorders: hypo-sodium, hypo-potassium, severe hyper-sodium, hypophosphatemia, hypothyroidism or hyperthyroidism, diabetic non-ketogenic hyperosmolar coma, diabetic ketoacidosis, pheochromocytoma, etc. Some genetic diseases: glycogen accumulation disease, congenital phosphofructokinase deficiency, etc. Connective tissue disease: multiple myositis, sicca syndrome, etc.

In this case, the patient probably got RM as a result of severe craniocerebral trauma and hypernatremia. After standardized evaluation of brain death, the patient met the criteria for brain death and was in the state of brain death. Patients with brain death have a relatively high probability of RM. This has an important impact on the quality of organ donations.

Mechanism of acute kidney injury by RM: Oxidative stress caused by heme, a degradation product of myoglobin, which would cause severe oxidative damage to the kidney. In addition, myoglobin in the patient's blood exceeds the binding capacity of plasma globulin, and deposits in the glomerular filtration fluid obstruct renal tubule, leading to renal injury (as shown in Figure 3).[8] In brain-dead donors, RM would darken the kidney in extreme cases , severely damaging donor renal function. Therefore, it might cause donor renal abandonment or DGF after renal transplantation.

Therefore, timely treatment of RM of brain-dead donors is very important, which can alleviate the current organ shortage of donors to a certain extent. Currently, the generally recognized principle of RM treatment is to remove the etiology as soon as possible along with fluid replenishment and alkalized urine treatment as early as possible to prevent and treat serious complications. The key to the treatment is early stage fluid resuscitation, correction of low blood volume, and prevention of acute tubular necrosis. In recent years, continuous renal replacement therapy (CRRT) is the preferred for the treatment and prevention of AKI induced by RM, despite some controversies over intervention timing, treatment mode and dose. Once RM is diagnosed, especially with

renal injury, CRRT treatment should be conducted as soon as possible. In case of urine pH<5.6, myoglobin entering renal tubules would degrade into ferritin and heme that are toxic to renal tubules epithelial cells. Meanwhile, a large number of myoglobin tubules block renal tubules, causing AKI. Alkalized urine combined with CRRT treatment can significantly remove myoglobin from blood. The application timing of CRRT can directly affect the survival rate of patients. For this reason, it should be performed as early as possible, especially before early renal failure. Timely and active CRRT can play a key role in the rescue of patients.

In this patient, a large amount of myoglobin was removed through alkalinized urine, fluid replenishment, plasma exchange and CRRT. These efforts gradually unblocked renal tubular, providing a good recovery of renal function. It was the appropriate timing of treatment that ensured smooth transplantation operation and postoperative recovery of renal function.

CONCLUSION

Brain death donors are prone to rhabdomyolysis, so early diagnosis and treatment of rhabdomyolysis should be emphasized. Through timely treatment, myoglobin can be removed from the donor to improve the success rate of donor kidney donation and the recovery of renal function after transplantation.

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REFERENCES

1. Nimmo GR, Lambie AT, Cumming AD. Rhabdomyolysis and acute renal failure. Inten Car Med.1989;15(8):486-7.
2. Vanholder R, Sever MS, Erek E. Rhabdomyolysis. J Am Soc Nephrol. 2000;11(8):1553-61.
3. Mekeel KL, Moss AA, Mulligan DC. Deceased donor kidney transplantation from donors with acute renal failure due to rhabdomyolysis. Am J Trans. 2009;9(7):1666-70.
4. Better OS, Abassi ZA. Early fluid resuscitation in patients with rhabdomyolysis. Nat Rev Nephrol. 2011;7(7):416-22.
5. Brain Injury Evaluation Quality Control Centre of National H, Family Planning C. Criteria and practical guidance for determination of brain death in adults (BQCC version). Chin Med J (Engl) 2013;126:4786-90.
6. Petejova N, Martinek A. Acute kidney injury due to rhabdomyolysis and renal replacement therapy: a critical review. Crit Care. 2014;18(3):224.
7. Cervellin G, Comelli I, Benatti M. Non-traumatic rhabdomyolysis: Background, laboratory features, and acute clinical management. Clin Biochem. 2017;50(12):656-62.
8. Chen X, Huang G, Yang S. Successful Transplantation of Black Kidneys Due to Myoglobin Nephropathy. Transplant Proc. 2018;50(8):2436-8.

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