

## CASE REPORT

# A case of rhabdomyolysis after kidney transplantation successfully managed with intensive continuous dialysis

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## Abstract

Rhabdomyolysis is characterized by muscle cell death which can result in acute kidney injury from pigment nephropathy. We present a patient who developed rhabdomyolysis immediately after deceased donor kidney transplantation surgery and was managed with continuous renal replacement therapy that resulted in successful salvage of the kidney allograft. Patients who develop acute kidney failure requiring renal replacement therapy generally have a poor prognosis. It is worth noting that while continuous veno-venous hemofiltration (CVVHF) offers greater volume support and continuous clearance compared to hemodialysis (HD), recent studies have demonstrated no clinically significant improvement in clinical outcome between the two. Perhaps CVVHF is a better modality compared to HD in this setting to prevent further insult from pigment nephropathy to an allograft. A combination of early diagnosis and intensive continuous renal replacement therapy can be used for allograft salvage in a patient with rhabdomyolysis in the immediate post-kidney transplant period.

## INTRODUCTION

Rhabdomyolysis, the destruction of myocytes, is one of many intrinsic insults to the kidney that can cause acute kidney injury (AKI) [1]. External injury (trauma, frostbite, muscle compression, prolonged immobilization), genetic or metabolic myopathies, inflammatory myopathies, infectious diseases, electrolyte abnormalities, hyperthermia, drugs and toxins have all been shown to cause rhabdomyolysis [2]; <10% of cases have no identifiable cause. Mechanisms underlying the pathogenesis of rhabdomyolysis are not clear [3]. Myoglobin and

creatinine kinase (CK) are released from the damaged muscle, freely filtered by glomeruli, and subsequently cause tubular damage by precipitating as casts and generating free radicals [4]. Myoglobin has also been shown to cause ischemic damage by inducing vasoconstriction of renal arterioles [5]. Hypovolemia resulting from influx of extracellular fluid into damaged muscle and hyperuricemia increase the risk of AKI. Rhabdomyolysis can also result in hyperphosphatemia, hypocalcemia and hyperkalemia. More than 15% of cases of rhabdomyolysis have been reported to be complicated by AKI. Herein, we present a patient

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who developed rhabdomyolysis immediately after kidney transplantation surgery. The allograft was salvaged through a combination of early diagnosis and intensive continuous renal replacement therapy.

## CASE REPORT

We present the case of a 38-year-old African American male with history of live kidney donation 12 years prior to his twin brother, for focal segmental glomerulosclerosis (FSGS) nephropathy, who subsequently developed biopsy-proven FSGS in the remaining kidney. This progressed to end stage kidney disease (ESKD). At the time of deceased donor kidney transplant, the patient was anuric and had been hemodialysis (HD) dependent for over 6 years. He did not receive any statins before surgery.

Deceased donor kidney transplantation was performed utilizing induction immunosuppression with methylprednisolone and antithymocyte globulin. He did not receive calcineurin inhibitors (cyclosporine, tacrolimus) or antimetabolites during the perioperative period. The intraoperative course was complicated by moderate hypotension, requiring pressor support. His post-operative blood chemistry showed serum potassium of 7.2 meq/L with hyperphosphatemia and hypocalcemia. To manage electrolytes, the patient underwent urgent HD. He was extubated on post-operative day (POD) 1 and was dialyzed again for hyperkalemia and hyperphosphatemia. He remained anuric and complained of paresthesias in bilateral hands and feet and pain in both thighs. With this triad of pain, hyperkalemia and hyperphosphatemia, rhabdomyolysis was suspected. Creatine kinase (CK) level was 41 000 U/L. Continuous venovenous hemofiltration (CVVHF) was initiated with blood flow rate 350 mL/h and high replacement fluid rate of 3500 mL/h to augment convective clearance of myoglobin and protect the kidney allograft from pigment nephropathy. On POD 2, CK peaked at 50 000 U/L and was cleared over the next 5 days. The patient was weaned off CVVHF to intermittent HD on POD 6 when CK level had decreased to 7500 U/L. By POD 8 the patient's CK was down to 3903 U/L and he was deemed ready for discharge. His kidney function and urine output continued to improve over the coming weeks; by POD 28 his creatinine had decreased to 1.7 mg/dL and CK was within the reference range. The patient has maintained stable kidney allograft function for 6 months without any further adverse events related to the transplant.

## DISCUSSION

We report the successful management of post-operative rhabdomyolysis with CVVHF in a patient who underwent deceased donor kidney transplantation associated with delayed graft function (DGF). While rhabdomyolysis was first described as a consequence of traumatic muscle injury, non-traumatic etiologies of rhabdomyolysis are now estimated to outnumber those caused by physical trauma [6]. Many intraoperative conditions can lead to skeletal muscle ischemia, including prolonged immobilization, tight dressings, malignant hypertension and vasospasm [7,8]. We believe that prolonged immobilization during the transplant procedure (7 h) combined with intraoperative hypotension in the setting of obesity (BMI = 37) could have contributed to the development of rhabdomyolysis in this patient. The intraoperative hypotension requiring vasopressor support may have led to vasospasm, muscle ischemia, and consequently rhabdomyolysis.

Elevated CK is the most sensitive measure for diagnosing rhabdomyolysis [9]. CK concentrations usually peak within the first 24 h of muscle injury [6]. The degree of CK elevation does not always predict the incidence of AKI [10]; however, AKI is uncommon when peak CK values are below 15 000 IU/L.

Initial treatment for rhabdomyolysis typically involves aggressive isotonic fluid administration to facilitate myoglobin clearance, but this method requires adequate kidney function to clear pigment and associated intracellular contents in the urine. This was impossible in an anuric patient with DGF. Thus, CVVHF was chosen for convective clearance of myoglobin to prevent further insult to the kidney allograft. With this, we were also able to manage hyperkalemia and hyperphosphatemia and optimize his volume status.

Patients who develop acute kidney failure requiring renal replacement therapy generally have a poor prognosis amounting to a 6–8-fold increase in mortality. It is worth noting that while CVVHF offers greater volume support and continuous clearance compared to HD, recent studies have demonstrated no clinically significant improvement in clinical outcome between the two. However, to our knowledge, this has not been studied in kidney transplant patients with DGF who develop rhabdomyolysis. Perhaps CVVHF is a better modality compared to HD in this setting to prevent further insult from pigment nephropathy to an allograft with DGF. Further studies are needed to investigate potential differences in outcomes based on the modality of renal replacement therapy used.

## CONCLUSION

This report demonstrates that a combination of early diagnosis and intensive continuous renal replacement therapy can be used to attempt allograft salvage in a patient with rhabdomyolysis in the immediate post-kidney transplant period.

## CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest regarding the publication of this article.

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