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EXTRACORPOREAL BLOOD PURIFICATION (HA-380) IN RHABDOMYOLYSIS-INDUCED ACUTE KIDNEY INJURY: A SUCCESSFUL OUTCOME

Marisa Cunha[1], Sílvia Sousa[1], Ricardo Marinho[1], Ana Castro[2], Aníbal Marinho[1]

[1] Intensive Care Unit, Unidade Local de Saúde de Santo António, Oporto, Portugal
[2] Nephrology Department, Unidade Local de Saúde de Santo António, Oporto, Portugal

Corresponding author's e-mail: anamarisa_c@hotmail.com

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ABSTRACT

Background: Rhabdomyolysis (RM) is a multifactorial syndrome characterised by skeletal muscle breakdown and release of intracellular components, notably myoglobin, into the circulation. Elevated myoglobin levels are a major contributor to acute kidney injury (AKI), yet conventional renal replacement therapies (RRT) are limited in their ability to clear myoglobin due to its molecular size.

Case description: We present a case of a woman who developed severe RM-induced AKI secondary to necrotising myopathy. Despite initial fluid and diuretic therapy, her condition progressed to oliguric AKI. She was admitted to intensive care unit and prolonged intermittent RRT was then initiated. Due to markedly elevated myoglobin levels, a haemoadsorption device (HA-380 hemoperfusion cartridge) was added to the RRT circuit, leading to significant laboratory and clinical improvement.

Discussion: Severe RM frequently leads to AKI due to myoglobin-induced tubular damage. While conventional RRT removes myoglobin poorly, haemoadsorption using the HA-380 cartridge effectively reduced plasma myoglobin and creatine phosphokinase levels in our patient, supporting early implementation to enhance renal recovery. Evidence from case reports and recent studies suggests safety and efficacy, but clinical outcomes remain incompletely defined, and optimal therapy timing, thresholds and duration require further investigation.

Conclusion: This case highlights the potential utility of extracorporeal adsorption therapy as an adjunct to RRT for effective myoglobin removal in severe RM-induced AKI. Early implementation of this strategy may enhance renal recovery and improve outcomes, though clinical evidence remains limited.

KEYWORDS

Rhabdomyolysis, acute kidney injury, renal replacement therapy, blood purification, haemoadsorption

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LEARNING POINTS

- Rhabdomyolysis is characterised by muscle breakdown with release of creatine kinase and myoglobin into the bloodstream. Elevated myoglobin levels are a major contributor to acute kidney injury and potentially permanent kidney damage.
- Conventional renal replacement therapies alone have limited the ability to remove myoglobin due to its molecular size. Timely haemoadsorption may offer a practical solution, enhancing myoglobin clearance and improving outcomes.
- Prospective studies are needed to establish the optimal use and efficacy of haemoadsorption in rhabdomyolysis-induced acute kidney injury.

INTRODUCTION

Rhabdomyolysis (RM) is a multifactorial syndrome characterised by skeletal muscle damage and release of intracellular contents into the bloodstream. Key indicators of muscle damage include myoglobin, creatine phosphokinase (CK) and lactate dehydrogenase (LDH)[1]. Although there is no universally accepted diagnostic criterion for RM, a CK level exceeding 1,000 IU/l or a five-fold increase above the upper normal limit (according to a local laboratory) is commonly used as a surrogate for diagnosis[2].

RM can result from various conditions and factors, including traumatic muscle injury, extreme physical exertion, infections and sepsis, metabolic myopathies, drug intoxications and side effects of toxins[3]. Clinically, RM can range from mild symptoms, such as myalgia and muscle oedema, to severe complications such as acute kidney injury (AKI), which may require organ support. Approximately 10–40% of RM cases lead to AKI, and 5–10% of AKI cases are due to RM[4].

Myoglobin, an iron-containing protein with a molecular weight of approximately 17 kDa, plays a central role in RM-induced AKI, due to its direct toxic effect on the tubule cells[1,4,5]. Excessive myoglobin accumulation may exceed the kidneys' clearance capacity, leading to persistent and potential permanent damage to tubular epithelial cells – significantly increasing the mortality rate, which can reach up to 80% in AKI patients[5]. Therefore, restricting the damage by an early and effective approach to myoglobin removal is critical to prevent or, at least, mitigate the AKI development.

Renal replacement therapy (RRT), particularly when AKI is already established or when an urgent indication for RRT exists, is conventionally used to remove myoglobin from the bloodstream[5]. However, due to its molecular weight, myoglobin is only poorly removed through conventional extracorporeal techniques[4]. High permeability membranes and high cut-off dialysers have been described as solutions for a better myoglobin removal, yet their use has also been associated with loss of albumin and coagulation factors[5,6].

An alternative approach towards myoglobin clearance is the use of haemoadsorption technologies. Used in pro-inflammatory states to control and remove inflammatory cytokines, a growing body of evidence is now supporting adjuvant adsorption therapy as an effective method to reduce elevated circulating levels of myoglobin, with clinical and laboratorial improvements, as well as shortening the intensive care unit (ICU) and hospital length of stay[7,8]. Nevertheless, the quality of the evidence is still poor, so there is a need for more data with clearly defined and relevant outcomes. An HA-380 disposable haemoperfusion cartridge is filled with neutral microporous resin, capable of adsorbing molecules from 10 to 60 kDa, including myoglobin[9].

Aiming to improve the knowledge in this field, we present a case of severe RM-induced AKI, initially manifesting with nausea and vomiting, lower limb pain and decreased urinary output, managed with a haemoadsorption cartridge (HA-380) added to RRT circuit, leading to the patient's successful recovery.

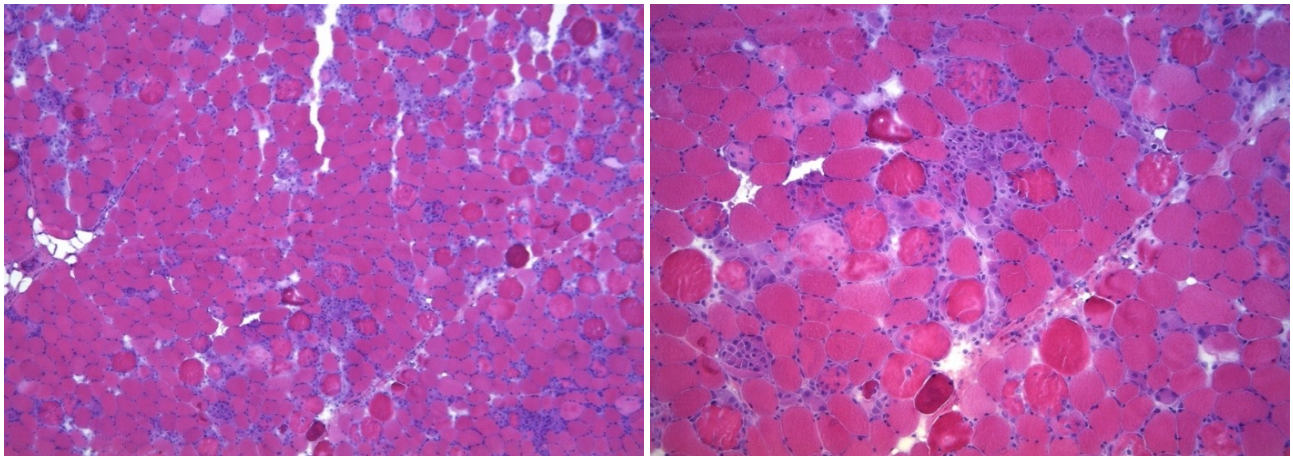


Figure 1. Haematoxylin and eosin colouration: images at different magnifications ×5 on the left and ×10 on the right – reveal numerous necrotic muscle fibres, along with scattered basophilic fibres within the tissue fragments. There is no perifascicular predominance, and no inflammatory infiltrates are observed. The higher-magnification image highlights muscle fibres at various stages of necrosis.

CASE DESCRIPTION

A female patient in her 30s came to our emergency department due to abdominal pain, nausea and vomiting lasting for four days. She also mentioned lower limb pain and an increased low back pain related to recent physical activity and decreased urinary output for approximately two days. She had a relevant medical history of obesity and chronic low back pain due to major surgery, evolving over approximately two years, for which she was being treated with nonsteroidal anti-inflammatory drugs. She denied taking other medications or drugs. She had no other significant medical, surgical, family or allergy history.

On admission, her physical examination revealed some discomfort on epigastric superficial palpation and muscle pain on both thighs. She was haemodynamically stable with no fever. The remainder of the physical examination was unremarkable.

As presented in *Table 1*, laboratory findings showed severe RM, indicated by myoglobin levels of 6,338 µg/l, CK levels >173,847 U/l and LDH levels of 4,106 U/l, plus an AKI reported by a serum creatinine value of 4.25 mg/dl in a patient with a previous normal renal function. The serum electrolyte panel was within normal range. Electrocardiography showed no signs of acute ischaemia, and an abdominal/renal-bladder ultrasound revealed no pathological findings.

Despite conservative therapy with fluids and diuretics, the patient's status did not improve, ultimately developing an oliguric kidney injury. She was then admitted to the ICU and prolonged intermittent RRT was initiated. Due to the markedly elevated myoglobin levels observed, a haemoadsorber (HA-380 HP cartridge, Jafron Biomedical Co.) was integrated into the RRT circuit. Overall, four sessions were performed, each lasting eight hours with a blood flow of 250 ml/min. The HA-380 cartridge was placed before the filter.

Myoglobin levels were lowered significantly, from 63,380 µg/l to 11,974 µg/l as well as CK levels, from >173,847 U/l to 119,833 U/l, within the first eight hours of prolonged intermittent RRT plus haemoadsorption. Kidney function, myoglobin and CK levels evolution can be seen in *Table 1*.

On the seventh day following ICU admission, the patient was discharged to the nephrology ward. A significant improvement in RM parameters was achieved as well as in renal function, with serum creatinine reaching normal values *(Table 1)*, plus urine output fully recovered.

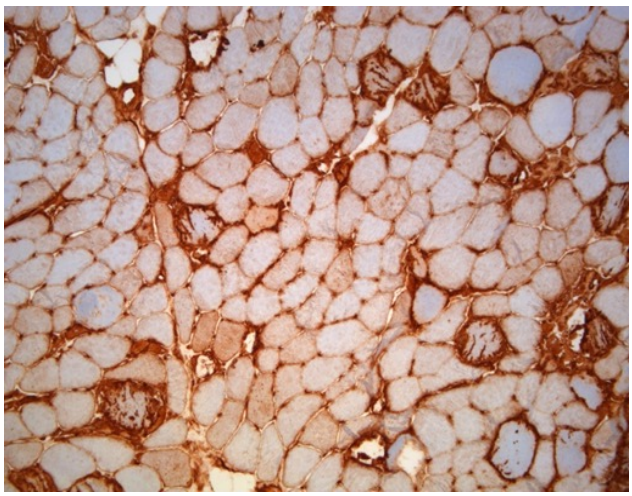


Figure 2. Major histocompatibility complex class I overexpression is evident in necrotic fibres, whereas the remaining muscle fibres exhibit weak to absent expression.

Throughout hospitalisation, an exhaustive investigation was performed to determine RM aetiology. The study of metabolic diseases and immunologic assay, including myositis studies, were unremarkable. Upper and lower limb electromyography found no signs of myopathy or polyneuropathy. Lower limb MRI showed oedema in both quadriceps, related to myositis.

As described in *Figure 1* and *Figure 2*, biopsy from quadriceps muscles was diagnostic for necrotising myopathy of uncertain aetiology. Intravenous immunoglobulins plus corticosteroids were then initiated.

Fifteen days after hospital admission, the patient was discharged home with fully recovered renal function and ongoing improvement in RM parameters *(Table 1)*.

DISCUSSION

Severe RM often leads to AKI, largely due to myoglobin-induced injury to proximal tubular epithelial cells. Myoglobin precipitates with Tamm–Horsfall protein, forming tubular casts, and triggers inflammatory pathways[5]. Persistent high myoglobin levels further amplify tubular damage and correlate with delayed renal recovery.

Although the rationale for conventional extracorporeal

	D0	D1	D2	D3	Discharge	Reference values
Myoglobin (µg/l)	63,387	11,974	2,235	2,908	112	25–58
CK (U/l)	>173,847	119,833	39,264	31,266	370.6	24–173
pCr (mg/dl)	4.25	1.63	0.76	0.80	0.81	0.5–0.9
Lactate (mmol/l)	0.5	0.6	0.9	1	0.9	0.5–2
LDH (U/l)	4,106	2,438	950	955	354	135–214

Abbreviations: CK, creatine phosphokinase; LDH, lactate dehydrogenase; pCr, plasmatic creatinine; D, day.

Table 1. Timeline values of the clinical case.

purification techniques is strong, evidence remains inconclusive resulting in limited clinical impact, as standard dialysis membranes remove myoglobin poorly[4]. High cut-off filters can enhance myoglobin clearance but require large fluid volumes and carry risks of albumin and coagulation factor loss[4,6].

All therapeutic options should be considered to prevent kidney crush syndrome; however, AKI is often unavoidable, particularly with high myoglobin levels. Haemoadsorption has a role in current critical care practice – while much attention has focused on its use in sepsis, these therapies have been effective in removing toxins and drugs[9]. Myoglobin clearance remains a potential therapeutic strategy although its safety, feasibility and efficacy are still under investigation. Our case demonstrates that use of the HA-380 haemoadsorption cartridge in this patient with severe RM successfully reduced plasma myoglobin and CK levels. Based on these findings, we strongly support early implementation of this adsorber to enhance myoglobin removal from circulation. Several case reports corroborate our observations, describing renal recovery evidenced by progressive decreases in urea and creatinine, increased urine output and/or cessation of RRT[7,8,10]. A recent retrospective propensity-matched study of 95 patients with severe RM and myoglobin levels >10,000 ng/ml, undergoing continuous RRT with (n=55) or without (n=40) adsorption therapy, reported significantly higher kidney recovery in the adsorption-treated group (31.4% vs 11.4%, p=0.04)[11].

Current literature suggests that haemoadsorption technologies efficiently remove myoglobin, reduce circulating CK levels and may support clinical improvement, with no severe device-related adverse events reported. Mild, asymptomatic thrombocytopenia has been occasionally described, requiring no intervention[12].

The most consistent effect of RRT combined with haemoadsorption in RM is myoglobin reduction. However, as highlighted by Forni et al., although results are promising, evidence regarding clinical outcomes remains limited and inconclusive[13].

Optimal timing for therapy initiation, relevant biochemical thresholds, adsorber capacity, monitoring strategies and therapy duration remain undefined. Larger prospective studies are needed to provide more robust data and clarify the role of haemoadsorption in RM-induced AKI.

CONCLUSION

Given the main role of myoglobin in the development of RM-induced AKI, it follows that early and effective reductions in elevated levels may prevent the development of AKI and its further consequences or at least shorten its duration. In our patient, RRT combined with HA led to effective myoglobin clearance in a severe RM-induced AKI case. This case highlights that early implementation of this strategy may enhance renal recovery and improve outcomes, though further prospective studies are needed to establish its efficacy and optimal use in clinical practice.

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