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Case Report

CytoSorb hemoadsorption in rhabdomyolysis-induced acute kidney injury: a case report from a tertiary hospital in Bangladesh

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ABSTRACT

Rhabdomyolysis is a serious clinical condition involving the breakdown of skeletal muscle, leading to the release of intracellular components into circulation. Among its complications, acute kidney injury (AKI) is the most critical, occurring in up to 50% of cases. This report presents the case of a 42-year-old male with schizophrenia and hypertension who developed rhabdomyolysis-induced AKI following a fall. He exhibited markedly elevated creatine phosphokinase (CPK) levels (360,000 u/l) and deteriorating renal function. In addition to supportive management, CytoSorb hemoadsorption was initiated. Over three sessions, CPK levels dropped to 174 u/l, and serum creatinine declined from 3.53 mg/dl to 2.16 mg/dl. This case demonstrates that CytoSorb therapy can be effective even in limited-resource settings where myoglobin assays are unavailable, offering a promising strategy for early intervention in rhabdomyolysis-induced AKI. Our experience suggests early initiation of hemoadsorption could serve as an effective adjunct in managing challenging cases where conventional therapies alone may be insufficient.

Keywords: Rhabdomyolysis, CytoSorb, Acute kidney injury, Hemoadsorption, Creatine phosphokinase

INTRODUCTION

Rhabdomyolysis is a life-threatening syndrome characterized by the release of intracellular contents such as myoglobin, potassium, phosphate, creatine kinase (CK), and lactate dehydrogenase into the bloodstream following skeletal muscle breakdown.[1,2] This cascade often results in acute kidney injury (AKI), primarily due to the

nephrotoxic effects of myoglobin and secondary oxidative tubular injury, renal vasoconstriction, and tubular obstruction.[3,4] Several risk factors, including trauma, seizures, drug use, and metabolic disorders, contribute to the onset of rhabdomyolysis. High serum CK levels, typically >15,000 u/l, are significantly associated with the development of AKI.[5,6] Management involves aggressive hydration, forced diuresis, and, when necessary, renal replacement therapy (RRT).[7]

In recent years, extracorporeal blood purification techniques such as CytoSorb hemoadsorption have gained prominence due to their ability to remove molecules up to 55 kDa, including myoglobin and inflammatory cytokines.[8,9] Unlike conventional dialysis membranes, CytoSorb efficiently adsorbs middle-sized toxins, making it ideal for severe rhabdomyolysis cases complicated by AKI.[10,11] Although most evidence comes from high-resource settings, emerging case reports indicate that CytoSorb may be applicable and beneficial in resource-limited hospitals as well.[12] Here, we present a case from Bangladesh to reinforce this therapeutic potential.

CASE REPORT

A 42-year-old Bangladeshi male with a past medical history of hypertension and schizophrenia-managed with long-term antipsychotic medications-was brought to the emergency department of a tertiary hospital in Chittagong, Bangladesh after a sudden fall at home approximately four hours prior to admission. On arrival, he presented with symptoms of agitation, drowsiness, and confusion. Clinical examination revealed bruises and abrasions on the back of the chest and forearm. His Glasgow coma scale (GCS) was E3M5V4=12. Vitals were stable initially. He was promptly stabilized and admitted to the High Dependency Unit (HDU) for close observation.

Initial laboratory investigations revealed a baseline serum creatinine of 0.77 mg/dl. However, within two days, his creatinine increased to 1.42 mg/dl, and the very next day it further escalated to 2.02 mg/dl, accompanied by reduced urine output (<1 ml/kg/hour), raising concern for acute kidney injury (AKI). Given the clinical context and the visible soft tissue injuries, rhabdomyolysis was suspected. Serum creatine phosphokinase (CPK) was sent and reported at a critically high level of 360,000 u/l, confirming the diagnosis. Due to unavailability of myoglobin assays, CPK served as a surrogate biomarker for muscle breakdown severity.

Considering the rapid renal deterioration and extreme hyper-CPKemia, CytoSorb hemoadsorption therapy was initiated alongside supportive care, including intravenous fluids and renal monitoring. A total of three CytoSorb sessions were conducted within 5 days. During therapy, creatinine peaked at 3.53 mg/dl but began declining afterwards, reaching 2.16 mg/d by the final session. CPK values showed a marked decline across sessions—dropping to 231,900 u/l, 162,200 u/l, and 10,259 u/l respectively—before normalizing to 174 u/l at discharge.

The patient remained in ICU for 9 days, was shifted to the ward on the 10th day, and was discharged on the 21st day post-admission with stable vitals, improved renal function, and no residual neurological deficits.

Results of laboratory parameters

Initially the serum CPK level in this patient was extremely elevated at 360,000 u/l, well above the critical threshold of 15,000 u/l commonly associated with rhabdomyolysis-induced acute kidney injury (AKI).[6] This severe hyper-CKemia reflects extensive skeletal muscle damage, a key diagnostic feature of rhabdomyolysis. Following the initiation of CytoSorb hemoadsorption therapy, there was a consistent and progressive reduction in CPK levels. After the first session the CPK level dropped to 231,900 u/l-indicating effective clearance of circulating muscle enzymes. The second session further reduced the value to 162,200 u/l, and by the third session, the CPK fell dramatically to 10,259 u/l-representing a nearly 97% decline from the initial value. At the time of discharge, the CPK had normalized to 174 u/l, indicating complete resolution of muscle breakdown (Table 1).

This downward trend confirms the effectiveness of CytoSorb therapy in clearing muscle breakdown products, despite the absence of direct myoglobin monitoring. The consistent reduction also suggests that early intervention with hemoadsorption can significantly reduce the biochemical burden, potentially minimizing further renal insult and systemic complications.

Table 1: Serum CPK levels during CytoSorb therapy.

Treatment stage	CPK level (u/l)
Before CytoSorb	360,000
After 1st session	231,900
After 2nd session	162,200
After 3rd session	10,259
Before discharge	174

Initially the patient's baseline serum creatinine was 0.77 mg/d which increased to 1.42 mg/dl within two days, at day 4 it rose further to 2.02 mg/dl. This rising trend of creatinine—combined with oliguria and elevated CPK—was indicative of evolving AKI secondary to rhabdomyolysis. During CytoSorb therapy, the serum creatinine level peaked at 3.53 mg/dl, suggesting the acute progression of renal impairment likely due to myoglobin-induced nephrotoxicity. However, after the full course of CytoSorb therapy, the creatinine began to decline, falling to 2.16 mg/dl by the end of the third session (Table 2). This reversal indicates a positive renal response to therapy, possibly due to decreased circulating toxins and reduced tubular injury. Although creatinine levels did not immediately return to baseline, the observed decline aligns

with renal recovery patterns described in similar clinical scenarios.[3,4,8] The trend highlights CytoSorb's adjunctive role in halting further renal deterioration and promoting early renal recovery.

Table 2: Serum Creatinine during hospital stay.

Stage of stay	Creatinine (mg/dl)
Admission	0.77
Day 3	1.42
Day 4	2.02
During CytoSorb	↑ 3.53 ↓ 2.16

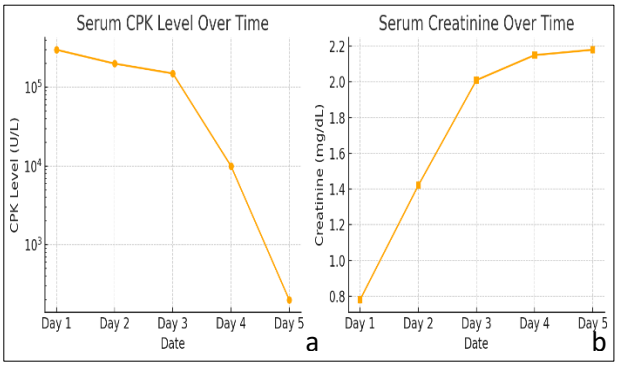

Figure 1 (a and b): Serum CPK and creatinine over time.

DISCUSSION

This case reinforces the utility of CytoSorb hemoadsorption in managing rhabdomyolysis-induced AKI, especially where traditional myoglobin detection is not feasible. CytoSorb can clear myoglobin and CPK effectively, aligning with studies that demonstrated significant reductions in these markers following treatment.[8,13] In their 2025 study, Pieri et al described three critically ill patients with rhabdomyolysis, all showing clinical improvement following dual CytoSorb therapy.[8] While our patient received single-filter therapy, the outcomes remain consistent with the observed benefits in CPK reduction and renal function improvement. This suggests that even a simplified CytoSorb protocol can yield considerable benefits. Safari et al emphasized that CPK levels are reliable predictors of AKI in rhabdomyolysis, particularly when levels exceed 15,000 u/l.[6] Our patient's baseline of 360,000 u/l was far beyond that, indicating high nephrotoxicity risk. The gradual decline of CPK post-treatment supports the role of CytoSorb in mitigating this risk. Albrecht et al and Dilken et al also highlighted CytoSorb's effectiveness in rapidly lowering CPK and myoglobin, echoing our findings.[9,10] Additionally, Graf et al underlined the importance of cartridge saturation kinetics, recommending multiple sessions or dual cartridges to maintain optimal toxin clearance.[13] While dual-cartridge setups were not used in this case, the single-cartridge approach still proved effective.

Our report is aligned with data from Heyne et al who described successful outcomes using high cut-off membranes, though CytoSorb has shown superior adsorption capacity.[11] In resource-limited settings, this case highlights CytoSorb's feasibility and clinical relevance, even without access to myoglobin assays.

CONCLUSION

This case report supports the use of CytoSorb hemoadsorption in severe rhabdomyolysis-induced AKI. Despite limited diagnostic facilities, a targeted therapeutic approach using CytoSorb led to significant clinical improvement, including reduction in CPK and stabilization of renal function. CytoSorb offers a practical, effective option for toxin clearance in critically ill patients, particularly in resource-constrained environments.

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