

Hemoadsorption with Cytosorb in AKI secondary to rhabdomyolysis induced by an experimental drug for Duchenne muscular dystrophy: A Case Report

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Introduction

The accumulation of myoglobin in renal tubules during acute rhabdomyolysis frequently results in acute kidney injury (AKI), often requiring initiation of kidney replacement therapy (KRT). Several studies have demonstrated that the use of the Cytosorb hemoadsorption device can enhance renal recovery in patients with severe rhabdomyolysis.

Methods

We report the case of a 14-year-old male patient with Duchenne muscular dystrophy who developed AKI secondary to rhabdomyolysis, likely associated with the administration of an experimental drug received as part of a clinical trial for Duchenne disease conducted at another institution. Four days after the infusion of the biologic agent, the patient developed vomiting, diarrhea, fever, and gross hematuria. After three additional days, due to reduced urine output and peripheral edema, he presented to the emergency department of our hospital.

Initial laboratory findings revealed AKI with a serum creatinine of 1.45 mg/dL and urea of 200 mg/dL, along with anemia, thrombocytopenia, elevated CPK levels (65,590 U/L), and markedly increased serum myoglobin (4,764 ng/mL).

Results

Due to clinical deterioration, the patient underwent two sessions of plasma exchange, multiple red blood cell and platelet transfusions, and three boluses of methylprednisolone (500 mg each). Given the suspicion of atypical hemolytic uremic syndrome (aHUS), treatment with the anti-C5 monoclonal antibody eculizumab was initiated. Simultaneously, intermittent continuous veno-venous hemodiafiltration (CVVHDF) was started (ultrafiltration rate: 30 mL/kg/h; session duration: 8–12 hours), in combination with the Cytosorb adsorption device.

Following the first treatment session, there was a marked improvement in the patient's clinical condition and a significant reduction in serum myoglobin levels (from 6,521 ng/mL to 4,601 ng/mL), which further improved after the second treatment (from 4,601 ng/mL to 1144 ng/mL) (Figure 1). CVVHDF sessions were discontinued after normalization of clinical and laboratory parameters. At discharge, renal function and myoglobin levels were within normal limits.

Conclusion

This case supports existing evidence that the use of Cytosorb in conjunction with KRT not only effectively removes circulating myoglobin but may also improve renal and overall clinical outcomes in patients with rhabdomyolysis-induced AKI.

Figure 1: myoglobin trend

