

Critical Illness Myopathy after septic shock in Pneumococcal pneumonia: Rhabdomyolysis treatment with Cytosorb cartridge

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Background

Critical Illness Myopathy (CIM) is a major complication in critically ill patients due to muscle weakness, energetic dysfunctions and many other factors. [1] Pro-inflammatory cytokines and metabolic imbalance as well as malnutrition and drugs are the trigger of these processes. [2] The severity of muscle weakness is widely variable and might be associated with symptoms of paralysis. Monitoring necrosis muscular enzymes levels (i.e. LDH, AST, ALT, Myoglobin, CK) is critical in order to understand the evolution of the myopathy. Blood purification adjuvant techniques might be useful to control their levels, eventually protecting at the same time renal function.

Case presentation

- Female patient, 68 years old, with a complex picture compatible with a suspected CIM resulting from a previous solved episode of septic shock in Pneumococcal pneumonia.
 - SAPS II score was 58, corresponding to a predicted mortality of 64%.
 - Anamnesis underlined arterial hypertension, chronic obstructive pulmonary disease (COPD), hypercholesterolemia, obesity and cannabis use.

Prior the admission in our Intensive Care Unit, the patient was treated in a peripheral hospital for a septic shock condition, presenting severe respiratory failure, cardiocirculatory dysfunction, oliguria and severe lactic and metabolic acidosis. Then the patient was transferred in our department, presenting an instable condition with right hemopneumothorax, severe hypoxemia (PaO₂/FiO₂ 150 mm Hg) and requiring noradrenaline administration (0.5 µg/kg/min).

In the next days, after thoracic drainage and bronchoscopy, hemodynamic stabilization was obtained and sedation suspended. The patient was awake and alert but developed severe tetraplegia with only elbow flexion and extension of the toes. At the same time, an increase in necrosis muscular enzymes and transaminases was observed.

Needle electromyography (EMG) testing highlighted a suspected myopathy without sign of denervation and muscle biopsy was prescript to understand the myopathy etiology once the patient recovered from the rhabdomyolysis.

On Day 12, in front of the persistent increase in muscle enzymes and limited renal function (diuresis <200 ml/day), blood purification with Cytosorb adsorber was started in combination of a CRRT treatment for myoglobin and CK removal. The treatment was performed for three consecutive cycles for total of 72 hours.

Results

The course of standard laboratory muscle markers and myoglobin levels are shown in Table 1.

At the end of the three Cytosorb treatment, we observed an important decrease in circulating myoglobin and CK levels, respectively 78% and 87 % (Figure 1). Moreover, muscle enzymes continued to progressively improve over the following days, until reaching normal levels. On Day 13, renal function restored with diuresis recovery (1500 ml/h), avoiding any further kidney impairment. A slow progressive increase in limb mobility was observed.

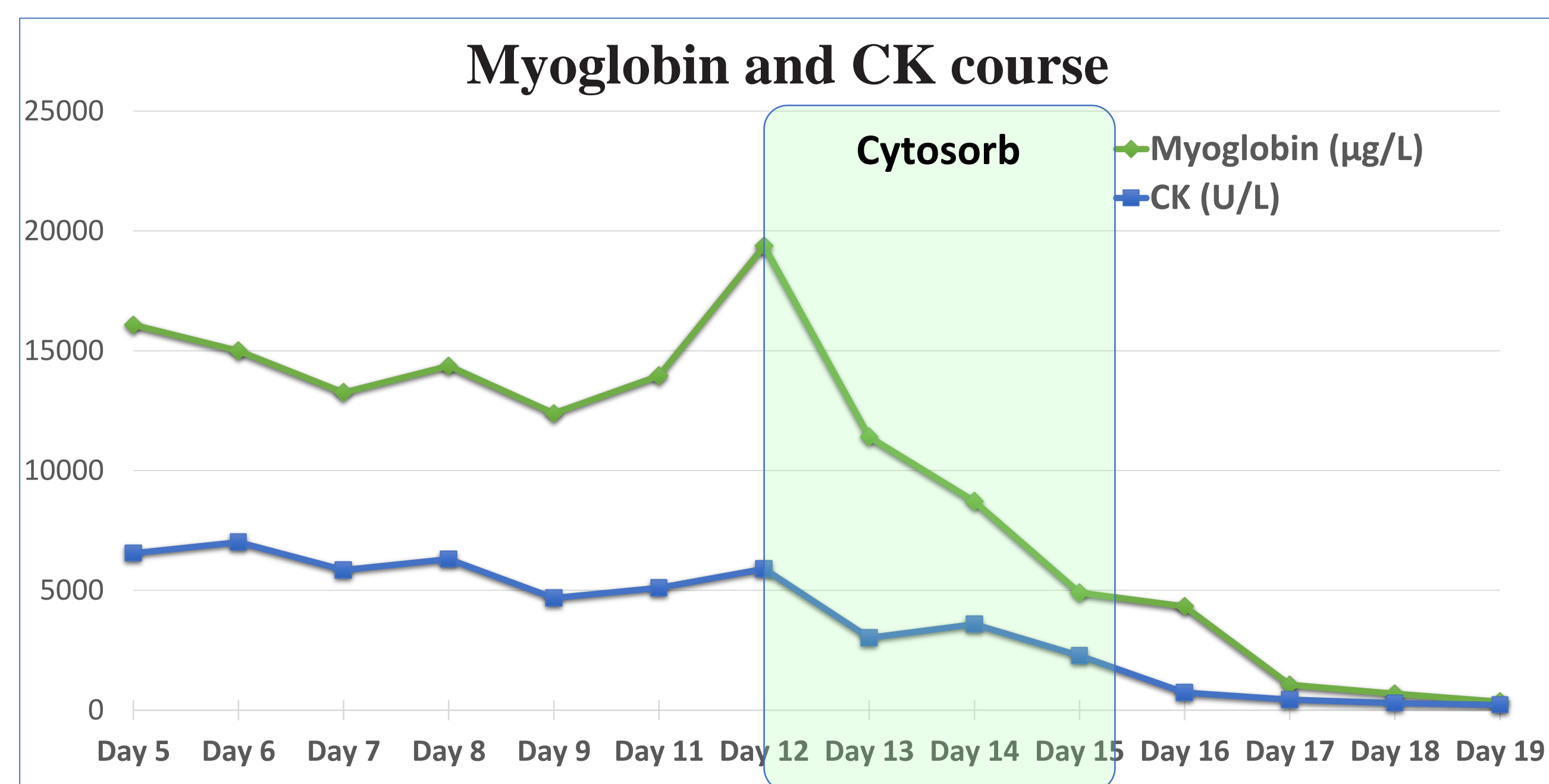


Figure 1: Myoglobin and CK course

	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13	Day 14	Day 15	Day 16	Day 17	Day 18	Day 19
Myoglobin (µg/L)	16,082	14,996	13,258	14,363	12,387	-	13,955	19,373	11,404	8,716	4,904	4,334	1,061	689	350
CK (U/L)	6,551	7,009	5,852	6,305	4,679	4,962	5,100	5,900	3,021	3,589	2,272	736	439	299	220
LDH (U/L)	409	424	394	406	477	599	-	628	655	880	968	919	866	-	-
AST/GOT (U/L)	322	365	335	362	336	390	-	382	320	382	305	184	141	125	99
ALT/GPT (U/L)	219	266	290	329	358	424	-	405	357	455	437	354	305	271	230

Table 1: Muscle enzymes and transaminase course

Conclusions

Cytosorb represents an easy support in case of rhabdomyolysis for a rapid removal of myoglobin and CK from blood and other muscle parameters stabilization, preventing at the same time further renal damages due to myoglobin nephrotoxicity.

References

1. Callahan LA et al. Sepsis-induced myopathy. Crit Care Med. 2009;37(10 Suppl):S354-67.
2. Rocheteau P et al. Sepsis induces long-term metabolic and mitochondrial muscle stem cell dysfunction amenable by mesenchymal stem cell therapy. Nat Commun. 2015;6:101.