Targeting shock and hyperinflammation with Cytosorb in patients supported with VA ECMO for refractory out-of-hospital cardiac arrest: a case-control study.

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Background

In patients resuscitated from refractory cardiac arrest, dysregulated iperacute inflammatory response is a deleterious clinical phenomenon which may be elicited from different clinical triggers, above all ischemia-reperfusion injury, contact with foreign surfaces during extracorporeal treatments, prolonged hemodynamic instability. Hemadsorption with Cytosorb (CytoSorbents Corporation, Monmouth Junction, NJ, USA) has given promising results in several clinical contexts. Its role in cardiac arrest patients, on the contrary, is debated and evidence contradictory. The aim of our study was to compare shock/inflammation laboratory parameters and outcomes of patients supported with extracorporeal cardiopulmonary resuscitation due to refractory (>60 min) out-of-hospital cardiac arrest treated with Cytosorb with a similar group of patients who didn't receive purification with Cytosorb.

Methods

Observational studies on all adult patients treated with VA ECMO for refractory cardiac arrest >48 hours at our institution from 4/2015 to 8/2020. Cytosorb was started after VA ECMO cannulation according to our institutional protocol whenever possible.

Results

One-hundred ten patients received VA ECMO >48 hours due to refractory cardiac arrest during the study period: 74 patients received Cytosorb treatment while 36 didn't receive Cytosorb. Cytosorb treatment duration was 46(24-63) hours. Shock and inflammatory parameters (baseline and peak values) for both groups are shown in table 1. All patients were on mechanical ventilation and extremely ill. Although patients in the Cytosorb group presented higher laboratory parameters of shock at baseline compared to patients not treated with Cytosorb (table 1), peak values of C reactive protein, total and direct bilirubin were not statistically different between the two groups (p=0.7, p=0.4 and p=0.8, respectively) Intensive care unit survival was 34/74(46%) in Cytosorb group and 20/36(56%) in no-Cytosorb patients (p=0.3); hospital survival was 34/74(46%) and 17/36(47%) in the same groups, respectively (p=0.9). No Cytosorb related adverse event was recorded.

Parameter at Baseline	Cytosorb group (n=73)	No Cytosorb group (n=36)	P value
Inotropic score	15 (8-27)	12 (6-20)	0.3
LDH U/L	934 (405 - 1660)	444 (368 – 656)	0.001
Troponin, ng/L	3040 (384 - 19294)	470 (191-2065)	0.002
CK U/L	2587 (401 - 7068)	343 (185-1448)	< 0.001
CRP, mg/L	16 (2-73)	1 (1-4)	0.001
Total bilirubin, mg/dl	0.6 (0.3 - 1.15)	0.3 (0.2-0.5)	< 0.001
Direct bilirubin, mg/dl	0.3 (0.12 - 0.54)	0.1 (0.1 – 0.2)	< 0.001
D-dimers, microgr/ml	19 (8-20)	9 (4-12)	0.01
Lactates, mmol/L	12 (8-18)	9 (7-11)	0.3
Parameter Peak			
Inotropic score	20 (14-32)	16 (10-30)	0.5
LDH U/L	1462 (840 – 2252)	895 (634 – 1235)	0.02
Troponin, ng/L	12657 (2136 – 25790)	4295 (2622 – 11713)	<0.001
CK U/L	6395 (2673 – 12445)	4159 (1258 – 7598)	0.04
CRP, mg/L	227 (141 – 310)	229 (139 – 272)	0.7
Total bilirubin, mg/dl	1.6 (0.9-2.3)	1.3 (0.7 – 2.8)	0.4
Direct bilirubin, mg/dl	0.8 (0.5-1.6)	0.7 (0.4 – 2)	0.8
D-dimers, microgr/ml	19 (10-20)	14 (10 – 18)	0.9
Lactates, mmol/L	11 (8-15)	9 (8-11)	0.07

Conclusion

We provided preliminary evidence that Cytosorb treatment is safe and effective in dumping blood circulation of molecules associated with shock and inflammation after cardiac arrest. Our data challenge existing data of possible increased mortality in patients treated with Cytosorb after cardiac arrest and highlights opportunities for further analysis in this setting.