



The use of CytoSorb extracorporeal hemadsorption in liver failure: a retrospective single-center registry study

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Background

Little is known about the use of CytoSorb[®] to provide biochemical control of liver impairment in patients with sepsis, acute on chronic liver failure (ACLF) or primary non function (PNF) after liver transplantation (LT).

Methods

This retrospective single-center study was conducted to elucidate beneficial and side effects of CytoSorb[®] in 17 consecutive patients treated during the period January 2020 to February 2022: 3 (18%) with liver failure during septic shock, 8 (47%) with PNF, and 6 (35%) with ACLF. All patients had received primary continuous renal replacement therapy prior to combination with CytoSorb[®]. The CytoSorb[®] effect was quantified comparing the following laboratory parameters pre-therapy and after 12-hour treatment periods: SOFA score, MELD score, CLIF-C ACLF, the need for inotropic drugs, mean arterial pressure, cardiac index, PaO₂/FiO₂, lactate, pH, procalcitonin levels, bilirubin, creatinine, BUN, platelet count, INR/PTT, transaminases, and IL-6.

Results

Mean/median bilirubin, blood urea, procalcitonin, proinflammatory cytokines and IL-6 levels fell significantly following treatment (Table 1). In contrast, no significant improvement was observed in hemodynamics, coagulation profile, and respiratory function. In-hospital ICU mortality of CytoSorb[®]-treated patients was 47%.

Conclusion

As a limited, non-granular, self-reported study, the only accurate outcome to demonstrate CytoSorb[®] efficacy was considered to be in-hospital mortality. The mortality reported for this series (47%) was on average lower than expected in light of the extremely poor patient scores at the time the decision was taken to start CytoSorb[®] treatment: 87.7% for SOFA score > 14; 52.6% for MELD ≥ 39; 100% at 3-7 days for CLIF-C ACLF > 65.

Although only registry data, our experience nonetheless shows a clear association between hemadsorption and lower predicted mortality. Extracorporeal hemadsorption therapy should therefore always be considered an adjuvant along with standard treatment in high mortality-risk patients. In addition, it would seem a promising therapy to buy time to transplantation in patients with ACLF.

A randomized controlled trial is needed to further evaluate the efficacy and indications for hemadsorption in this uncommon clinical setting.

Reference

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	Baseline	12 hours after treatment	P value
SOFA score	16,5 ± 3,4	19,2 ± 2,4	<0,001*
CLIF-C ACLF	69,2 ± 9,5	72,5 ± 7,8	n.s.
MELD	39 ± 6 (19-47)	41 ± 9 (15-53)	n.s.
Creatinine (mg/dL)	1,6 ± 0,8 (0,8-4,0)	1,5 ± 0,7 (0,6-2,9)	n.s.
Urea (mg/dL)	70,5 ± 49,8 (13-162)	49,9 ± 28,5 (11-109)	0,04*
Bilirubin (mg/dL)	14,3 ± 12,6 (1,5-40)	10,7 ± 8,5 (2,5-26,4)	0,015*
Platelets (109/L)	63,4 ± 24 (20-108)	41,1 ± 18,1 (8-76)	0,003*
INR	3,1 ± 0,8 (2,1-5,5)	4,1 ± 2,0 (1,6-10)	0,007*
ALT (U/L)	1688 ± 2088 (15-6433)	1093 ± 1235 (35-3980)	n.s.
AST (U/L)	2922 ± 2991 (63-7000)	1733 ± 2113 (103-7000)	n.s.
Ammonia (mcg/dL)	186,7 ± 53,8 (132-247)	106,3 ± 23 (81-136)	n.s.
Albumin (g/dL)	3,2 ± 0,5 (2,2-4,2)	2,9 ± 0,4 (2,3-3,8)	n.s.
MAP (mmHg)	63 ± 11 (47-93)	63 ± 11 (32-75)	n.s.
Cardiac Index	3,5 ± 0,9 (1,8-4,8)	3,3 ± 1,2 (2,1-6)	n.s.
Norepinephrine (mcg/kg/min)	0,45 ± 0,26 (0,08-0,9)	0,43 ± 0,25 (0,06-0,9)	n.s.
Lactate (mmol/L)	9,1 ± 7,7 (1,19-26)	8,6 ± 6,4 (1,87-20)	n.s.
pH	7,30 ± 0,16 (7,01-7,52)	7,33 ± 0,10 (7,16-7,49)	n.s.
PaO ₂ /FiO ₂ ratio	327 ± 267 (73-1143)	253 ± 268 (88-1203)	n.s.
Procalcitonin (ng/mL)	9,8 ± 14 (0,4-49)	5 ± 10 (0,2-37)	0,02*
IL-6	3521 ± 4090 (6414-629)	562 ± 205 (707-417)	<0,001*

Table 1: Biochemical and hemodynamic parameters before hemoperfusion treatment and 12 hours after treatment. SOFA score: Sepsis-related Organ Failure Assessment; CLIF-C ACLF: Chronic Liver failure Consortium Acute-on-Chronic Liver Failure; MELD: Model for End-stage Liver Disease; INR: international normalized ratio; ALT: Alanine Transaminase; AST: Aspartate aminotransferase; MAP: mean arterial pressure; IL-6: interleukin-6
n.s.: p-value > 0.05