Effect of Coagulation Factor Concentrates on Markers of Endothelial Cell Damage in Experimental Hemorrhagic Shock

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Abstract

Background: Plasma-based resuscitation showed protective effects on the endothelial glycocalyx compared with crystalloid resuscitation. There is paucity of data regarding the effect of coagulation factor concentrates (CFC) on the glycocalyx in hemorrhagic shock (HS). We hypothesized that colloid-based resuscitation supplemented with CFCs offers a therapeutic value to treat endothelial damage following HS.

Methods: Eighty-four rats were subjected to pressure-controlled (mean arterial pressure (MAP) 30-35 mm Hg) and lab-guided (targeted cutoff: lactate >2.2. mmol/L and base deficit > 5.5 mmol/L) HS. Animals were resuscitated with fresh frozen plasma (FFP), human albumin (HA) or Ringer's lactate (RL) and RL or HA supplemented with fibrinogen concentrate (FC) or prothrombin complex concentrate (PCC). Serum epinephrine and the following markers of endothelial damage were assessed at baseline and at the end-of-observation (120 min after shock was terminated): syndecan-1, heparan sulfate, and soluble vascular endothelial growth factor receptor 1 (sVEGFR 1).

Results: Resuscitation with FFP had no effect on sVEGFR1 compared with crystalloid-based resuscitation (FFP: 19.3 ng/mL vs. RL: 15.9 ng/mL; RL+FC: 19.7 ng/mL; RL+PCC: 18.9 ng/mL; n.s.). At the end-of-observation, syndecan-1 was similar among all groups. Interestingly, HA+FC treated animals displayed the highest syndecan-1 concentration (12.07 ng/mL). Resuscitation with FFP restored heparan sulfate back to baseline (baseline: 36 ng/mL vs. end-of-observation: 36 ng/mL).

Conclusion: The current study revealed that plasma-based resuscitation normalized circulating heparan sulfate but not syndecan-1. Co-administration of CFC had no further effect on glycocalyx shedding suggesting a lack of its therapeutic potential.