Autotransfusion of Hemothorax Blood Is Prothrombotic But Inhibits Platelet Aggregation: A Potentiator of DIC

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OBJECTIVES: In austere environments, autologous hemothorax salvage is a potential option for resuscitation. However, recent studies of pleural blood demonstrated clotting factor deficiencies with hypercoagulatory thromboelastographic (TEG) changes. Previous assumptions regarding the hemostatic value of this product appear to be erroneous, and the combinations of hyper- and hypo-coagulable changes are reminiscent of disseminated intravascular coagulation (DIC). We examined potential mechanisms of coagulopathy and the possible impact of transfusing salvaged blood by approximating ratios of pleural hemothorax-derived plasma to that of healthy subjects in an in vitro model of simple and massive transfusion.

METHODS: Hemothorax blood (HX) was obtained from 17 adult trauma patients under an Institutional Review Board (IRB) approved prospective observational protocol. Tube thoracostomy blood samples were removed at 1-4 hours after injury; the corresponding plasma (HXP) was isolated and frozen for analysis. The effect of HX on coagulation was assessed by reproducing likely ratios of HXP to healthy subject platelet rich plasma (PRP) after transfusion, as well as isolating HXP microparticles (MPs). Massive transfusion was defined as a 1:1 ratio of HXP to PRP and simple transfusion as 1:7. Samples were analyzed by thromboelastometry (ROTEM), platelet aggregometry (Multiplate), enzyme-linked immunoabsorbent assays (ELISA), and flow cytometry.

RESULTS: ROTEM analysis demonstrated that the HXP and HXP MPs decreased clotting time (CT), clotting formation time (CFT), and increased the alpha angle (α) irrespective of the degree of dilution. Additionally, both HXP and HXP MPs inhibited platelet aggregation in response to stimulation with the following agonists: collagen, Thrombin Receptor Agonist Peptide (TRAP), adenosine di-phosphate (ADP), and platelet activator thromboxane A2 (ASPI). Fibrin degradation products (FDP) and tissue factor (TF) were elevated in shed plasma samples. Conversely, Anti-thrombin III (ATIII) decreased in shed blood plasma samples. Phosphatidylserine-, platelet-, and red blood cell-derived MPs increased compared to control plasma samples.

CONCLUSION: Mixing of shed blood plasma with healthy donor plasma produced derangements in global hemostasis, platelet function and in secretion of markers of coagulopathy. Exposure to the microparticle pellet similarly produced similar findings. Alterations were consistent with a mixed consumptive coagulopathy in the presence of accelerated clot initiation. These findings suggest that transfusion of pleural shed blood may result in DIC-like changes. Possible mechanisms include increased tissue factor, presence of fibrin-degradation products, decreased anti-thrombin III and increased exposure to phosphatidylserine platelet MPs available within the shed plasma. Autologous transfusion from shed hemothorax blood may be harmful.