Low Viscoelastic Clot Strength, Platelet Transfusions, and Graft Dysfunction are Associated with Persistent Postoperative Ascites Following Liver Transplantation

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Introduction

Persistent ascites (PA) following liver transplantation (LT) is a postoperative complication associated with increased morbidity, prolonged hospitalization, and higher healthcare utilization. While PA has historically been attributed to residual portal hypertension, emerging data suggest endothelial dysfunction and platelet-mediated hemostasis play a role. Platelets contribute to endothelial integrity and vascular repair, and dysfunction in this process may exacerbate fluid leakage. We assessed the association of PA with platelet count and platelet function as measured by thrombelastography (TEG). We hypothesized PA is associated with platelet dysfunction as measured by clot strength via viscoelastic testing.

Methods

Liver transplant recipients were prospectively enrolled in an observational coagulation study. Age >18 years, Child-Pugh score ≥2, and deceased donor LT were inclusion criteria. Citrated blood assayed with thrombelastography was collected before incision through postoperative day (POD) 7. PA was defined as drain output >1L on POD7. Clot strength was assessed via TEG maximum amplitude (MA), and transfusion data collected. Univariate and multivariate analyses identified pre- and postoperative predictors of PA.

Results

One hundred fifty LT recipients were included. Median age was 53 years, 37% female, and median MELD was 24. PA occurred in 28% of patients and was associated with higher preoperative MELD-Na ($p=0.024$) and increased platelet transfusions ($p=0.006$). PA patients had significantly lower POD5 TEG MA ($p=0.008$), increased drain output, longer hospital stays (17 vs. 10 days, $p<0.001$), and higher intraabdominal infection rates (10% vs. 1%, $p=0.011$). A composite of preoperative platelet transfusions, POD5 MELD-Na >17, and POD5 TEG MA <40mm identified a stepwise increase in PA risk.

Conclusion

PA following LT is significantly associated with perioperative platelet transfusions, impaired clot strength, and graft dysfunction. These findings suggest platelet function contributes to postoperative ascites. Further evaluation of platelet transfusions to reduce high drain output is warranted, but potential benefits must be weighed against thrombosis risk.