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27. DETECTION OF EARLY ALLOGRAPH DYSFUNCTION AT 30 MINUTES OF REPERFUSION IN LIVER TRANSPLANTATION: AN INTRAOPERATIVE DIAGNOSTIC TOOL WITH REAL TIME ASSESSMENT OF GRAFT FUNCTION

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Background: Early allograph dysfunction (EAD) in liver transplantation is associated with decreased survival time of the graft and early mortality in the recipient. Despite EAD occurring in a quarter of patients, there are no effective treatments. This is partially attributable to the diagnosis being made seven days after surgery, in which a therapeutic window may have been missed. One of the many roles of the liver is to clear tissue plasminogen activator(tPA) from circulation. It is well documented that during the anhepatic phase of surgery tPA levels are elevated and often associated with increased fibrinolytic activity. Abundant receptors in the liver clear tPA, and a rapid correction of fibrinolysis upon graft reperfusion would be expected in a well-functioning organ. Therefore, we hypothesize that patients that fail to reduce fibrinolytic activity following graft reperfusion will have an increased rate of EAD.

Methods: Assessment of fibrinolysis in liver transplant recipients was quantified by the lysis at 30 minutes (LY30) after the clot reached maximum strength using Thrombelastography (TEG). TEGs were contrasted between the anhepatic phase of surgery and 30 minutes following graft reperfusion. It was anticipated that the majority of patients would develop hyperfibrinolysis during the anhepatic phase of surgery (LY30>3%). The 30 minutes reperfusion LY30 was subtracted from the anhepatic LY30 quantifying fibrinolytic changes (delta-LY30). EAD was defined on the existing definition in the literature using a combination of post-operative labs through post-operative day-7. A receive operating characteristic curve (ROC) was used to define the threshold of delta LY30 for predicting EAD using a Youden index. Patients with a low delta-LY30 based on this cut point were contrasted to patients with a delta LY30 higher than this point and stratified development of hyperfibrinolysis during surgery.

Results: Eighty liver transplant patients with a median MELD of 19 were included in the analysis. EAD occurred in 25% of patients and 55% of patients developed hyperfibrinolysis during the anhepatic phase of surgery. ROC curve analysis identified an inflection point of delta LY30 -5.3% as a risk factor for EAD. Low delta-LY30 was prevalent in 66% and 1/3rds of these patients developed hyperfibrinolysis. EAD occurred in 47% of these patients compared to 28% (low delta-LY30 and no hyperfibrinolysis) and 11% (hyperfibrinolysis large delta-LY30 p=0.037 Figure). Patients with low delta-LY30 and hyperfibrinolysis had longer warm ischemia times than the other cohorts (39min vs 34min and 31min p=0.014). No other donor or recipient variable were significantly different between cohorts including MELD (p=0.139), baseline coagulation (P>0.100 for all), and underlying liver disease process (p=0.166).

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Conclusion: Hyperfibrinolytic liver transplant recipients that fail to reduce fibrinolytic activity 30 minutes after graft reperfusion had an EAD rate that approached 50%, while recipients that failed to generate a fibrinolytic response during surgery also harbor a high rate of EAD. These data suggest that there are donor and recipient factors which can be identified intraoperatively to risk stratify patients for EAD, which will aid in understanding this pathophysiology to develop effective treatment strategies.