MO418 Figure 1: The Kaplan-Meier survival analysis using the log-rank test for comparing the hemofilter lifespan between regional citrate (RCA) and no anticoagulation (NA) regime.

**CONCLUSION:**
- The bleeding propensity was significantly higher (56.3% vs 25.4%, p = 0.002) in the NA group compared to the RCA group.
- The overall duration of bleeding (Mantel-Cox test) was significantly longer in RCA-CRRT, which suggested the benefit of RCA in reducing bleeding compared to NA-CRRT in the patients with a high risk of bleeding.

**RESULTS:**
- The duration of bleeding was significantly shorter (4.82 vs 2.80, p = 0.040) and the volume of bleeding was significantly lower (2.25 vs 1.28, p = 0.027) in the RCA group compared to the NA group.
- The length of hospital stay and mortality (36.24 vs 486.0, p = 0.001) was significantly lower in the RCA group.
- The difference in survival between the groups (v^2 = 3.789, p = 0.049) (Figure 1) was also significant.
- The ultrafiltration rate (2.79 vs 6, p = 0.016) was significantly higher in the NA group, no significant difference was found between the groups (37.5% vs 28.20%, p = 0.498).
- Although the prevalence of bleeding was higher in the NA group, no significant difference was found between the groups (37.5% vs 28.20%, p = 0.498).
- The overall citrate accumulation (CA) rate was 5.12% in the RCA group vs 2.25) rate was 5.12% in the RCA group.
- The overall citrate accumulation (CA) rate was 5.12% in the RCA group.

**PROTOCOL:**
- The study was conducted in a randomized controlled open prospective study.
- Statin naive patients were included in the study.
- The interventions were as follows:
  - Group 1: 40 mg atorvastatin 24 hours and 40 mg 2 hours before CT scans and 40 mg after.
  - Group 2: 40 mg after.
  - Group 3: Control group, no atorvastatin.

**RESULTS:**
- The incidence of CI-AKI was significantly lower in the atorvastatin-treated groups compared to the control group.
- Statin therapy significantly reduced the burden of CI-AKI after CT scanning in statin naive patients with cardiovascular diseases.
- The development of CI-AKI was significantly reduced in the atorvastatin-treated groups.

**CONCLUSION:**
- Statin therapy significantly reduces the burden of CI-AKI after CT scanning in statin naive patients with cardiovascular diseases.
- The study suggests that statin therapy may be a preventive measure against CI-AKI.

**ROLE OF IL-6 ON ACUTE KIDNEY INJURY (AKI) DEVELOPMENT AFTER LIVER TRANSPLANTATION**

**BACKGROUND AND AIMS:**
- Acute kidney injury (AKI) post-liver transplantation is a frequent complication with an incidence up to 70%, requiring renal replacement therapy in about 25% of transplant patients.
- AKI in patients with normal renal function is a recognized risk factor (FR) of chronic renal failure (CKD) de novo, associated with a 4.5 times greater mortality at 5 years post-transplant.
- Pathogenesis of AKI is multifactorial. Beyond the classical pre-transplant risk factors, the hypoxia of the graft and the ischemia-reperfusion injury (IRI) have recently been recognized to exert a pathogenetic role with specific mechanisms.
- It has been recently demonstrated in experimental setting that ischemic tissues put in place protective mechanisms in response to hypoxia aimed at increasing the release of oxygen with the activation of angiogenesis mediated by the expression of factors induced by hypoxia (HIF)-1-alpha.
- HIF-1-alfa has been shown to promote cell survival under hypoxic conditions by switching metabolism from oxidative to glycolytic, by affecting the production of ATP to prevent excessive mitochondrial generation of reactive oxygen species, by promoting secondary release of vascular endothelial growth factor (VEGF) and transforming growth factor-beta 1 (TGF-β1), with following activation of inflammatory cytokines responsible for systemic inflammatory response syndrome (SIRS).
- Tumor necrosis factor-α, IL-1 and IL-6 are the most important cytokines released in IRI and seem to play a pivotal role in the onset of AKI in SIRS and sepsis.
- The development of AKI after hypoxia/ischemia of the graft, as observed more frequently in the population of recipients from donors after circulatory death (DCD) compared to donation after brain death (DBD), confirms this pathogenetic mechanism.

**AIM:**
- The aim of the study is to evaluate AKI occurrence among liver transplanted patients and its relationship with IRI and cytokines systemic release.
BACKGROUND AND AIMS: Of 158 patients (females=36.1%, mean age 60.0 hours postcardiac catheterization. A patient for cardiac catheterization. Chronic renal failure. This low cost intervention could be considered when referring a patient for cardiac catheterization. The incidence of CIN, whereas it has the potential to mitigate the incidence of CIN in patients with chronic renal failure. RAAS blockade isn't associated with a significantly higher incidence of CIN, whereas it has the potential to mitigate the incidence of CIN in patients with chronic renal failure.

CONCLUSION: There was no significant difference between the two groups, RAAS blockade 'used' vs 'not used,' with respect to the incidence of AKI (51.3% vs 51.2%, p=0.903).

METHOD: Data of 78 patients (62 males, 79.5%) underwent liver transplantation (2007-2011) were retrieved.

RESULTS: The following clinical investigations were performed:

- AKI developed in 40 patients (51.3%), of these 30 patients developed AKI stage 1, 5 patients AKI stage 2 and 5 AKI stage 3. Patients with AKI stages 2&3 presented more deteriorated pre-tx liver function as suggested by higher MELD score (AKI2&3 20 [16-25] vs no AKI-AKI1 15 [11-18]; p=0.008), higher bilirubin (AKI2&3 5.7 mg/dl [3.5-10.8] vs no AKI-AKI1 2.8 mg/dl [1.2-4.9]; p=0.009) and INR (AKI2&3 1.9 [1.6-2.5] vs no AKI-AKI1 1.4 [1.3-1.6]; p<0.0001) at transplant, despite superior renal function (serum creatinine in AKI2&3 3.7 mg/dl [0.5-0.8] vs no AKI-AKI1 0.9 mg/dl [0.7-1.1]).

- Patients with AKI2&3 experienced greater ischemia-reperfusion injury based on functional recovery of the transplanted liver (higher peak AST in the first 7 days post-LT p=0.029, and higher peak ALT level p=0.062).

- The evaluation of IL-6 levels at transplant, 1- and 12-days post- LT were performed in 21 patients, 8 with AKI and 13 with no AKI.

AKI patients demonstrated a progressive increasing of IL-6 after liver transplantation (AKI 34.4.37.8-88.2 ng/ml vs no AKI 30.5-21.6-23.3 ng/ml).

CONCLUSION: Patients who experienced greater ischemia-reperfusion injury of the liver graft developed more frequently AKI. Patients with AKI experienced an increased release and circulation of IL-6, that probably is involved in AKI development with interesting implications in future therapy.