Non-selective beta-blockers (NSBBs) have played a key role in the prevention of portal hypertensive bleeding in patients with cirrhosis. However, recent studies have suggested that NSBB use is associated with decreased survival in patients with refractory ascites. The aim of this study is to evaluate any association between NSBB use and the incidence of acute kidney injury (AKI).

Methods: We used a nested case-control design from the cohort of liver transplant waitlist registrants at Mayo Clinic, Rochester, USA. Cases consisted of patients who developed AKI ≥ stage 2, defined by a 2-3 fold increase in serum creatinine compared to baseline. Each AKI patient was matched to a control, based on MELD-Na score, age at registration, baseline creatinine, and follow-up duration.

Results: Out of the total cohort of 2250 waitlist registrants, 202 patients met the criteria of AKI. The median follow-up duration was 20.3 (range: 3-201) months. When compared to matched controls without AKI, ascites (78.7% versus [vs.] 52.0%), non-white race (16.3% vs. 7.9%) and absence of malignancy (89.6% vs. 82.7%) were more commonly seen among the cases. In the univariate proportional hazard regression analysis, male sex, caucasian, malignancy, autoimmune etiology, high MELD and MELD-Na at baseline, and ascites were significantly associated with development of AKI. In multivariable analyses, the impact of NSBB on AKI incidence was different according to the presence of ascites: NSBB use in patients with ascites was significantly associated with development of AKI (hazard ratio [HR], 2.66; 95% confidence interval [CI], 1.47-4.82), while in patients without ascites, NSBB was protective (HR, 0.24; 95% confidence interval [CI], 0.09-0.66), after adjusting for MELD-Na at baseline, sex, race, etiology of cirrhosis and presence of liver cancer.

Conclusions: The use of NSBB increased the risk of AKI in cirrhotic patients with ascites, which likely contributes to increased mortality.

Keywords: Non-selective beta-blocker, Acute kidney injury, Cirrhosis

**PS-2-4**

**Serum Cystatin C is a Powerful Prognostic Indicator in Patients with Cirrhotic Ascites: A Multicenter Prospective Observational Study**

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Background: Because renal dysfunction is often accompanied by progression of liver dysfunction and hepatorenal syndrome (HRS) is one of the major causes of mortality, accurate assessment of renal function is very important for the assessment of patients with cirrhotic ascites. Although several studies suggested that cystatin C (CysC) level is more reliable than creatinine level, it is still unclear whether CysC could be useful as a prognostic marker in these patients. This study was performed to evaluate the clinical significance of CysC in patients with cirrhotic ascites.

Methods: Patients with cirrhotic ascites were prospectively enrolled between Sep 2009 and Mar 2013 at 14 hospitals. Patients with hepatocellular carcinoma or parenchymal kidney disease or those taking diuretics were excluded. Laboratory tests including serum creatinine and CysC were performed at the time of enrollment.

Results: Three-hundred forty-six patients were enrolled. Age was 55.3±11.0 years and 262 patients (75.7%) were male. The most frequent cause of liver disease was alcoholic liver disease (35.6%), followed by chronic hepatitis B (31.2%). Serum creatinine and CysC levels were 0.10±0.5 mg/dL and 1.1±0.5 mg/L, respectively. During 36.7±1.5 months of follow-up, 88 patients died. Cause of mortality was as follows: HRS, 27(7.8%), liver
failure, 26(7.5%); variceal bleeding, 13(3.8%); sepsis, 9(2.6%); others, 9(2.6%). Survival time was 36.7±1.5 months and 6- and 12-month survival rates were 86.7% and 80.8%, respectively. INR, bilirubin, albumin, glucose, BUN, creatinine, sodium, and CysC levels were significant factors on univariate analysis, while sodium and CysC levels and INR were independently associated factors with survival on multivariate analysis. The incidences of HRS at 6 and 12 months were 5.4% and 7.6%, respectively. Similarly, sodium and CysC levels and INR were independent factors for predicting development of HRS. 

Conclusions: Serum CysC level was a powerful indicator for mortality and development of HRS in patients with cirrhotic ascites.

Keywords: Liver cirrhosis, Ascites, Cystatin C, Hepatorenal syndrome, Prognosis

**PS-2-5**

**Effects of Probiotics (Cultured Lactobacillus Subtilis/Streptococcus Faecium) in the Treatment of Alcoholic Hepatitis: Randomized Controlled Multicenter Study**

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Background: Gut-derived microbial lipopolysaccharide (LPS) has been known as a central role in the pathogenesis of alcoholic hepatitis (AH). Some studies suggested an emerging role of probiotics in restoration of the bowel flora and improving liver enzymes. We evaluated the therapeutic effects of probiotics in patients with AH.

**Methods:** Between September 2010 and April 2012, 117 patients (probiotics: 60 and placebo: 57) were prospectively randomized to receive 7 days of cultured lactobacillus subtilis/streptococcus faecium (1,500 mg/day) or placebo. The mean levels of liver enzymes, mDF, LPS, pro-inflammatory cytokines, and stool culture were checked at baseline and again after therapy. AH was defined as an AST/ALT>1 and elevated AST(ALT) level with an alcohol consumption history within 48 hours.

**Result:** In the probiotics group, the mean level (baseline vs. after) of AST (236±606 vs. 66±41 IU/L), ALT (93±154 vs. 47±47 IU/L), alkaline phosphatase (ALP)(130±53 vs. 117±42 IU/L), gamma glutamyl-transferase (GGT)(474±584 vs. 306±398 IU/L), and mDF (24±54 vs. 16±33 IU/L) were improved. In the stool culture, CFU of gram negative bacteria was significantly reduced (435[287] vs. 168[210]). The level of interleukin 1β (51[85] vs. 43[83]), tumor necrosis factor (TNF)-α (121 [244] vs. 71 [123]), and LPS (1.8 [1.8] vs. 1.7 [1.6]) did not show difference. In the placebo group, levels of AST, ALT, ALP, and RGT were significantly improved after 7 days. The level of mDF (9 [21] vs. 8 [21]), interleukin 1β (37 [55] vs. 14 [6]), TNF-α (45 [54] vs. 34 [34]), and LPS (1.7 [2.8] vs. 2.0 [2.7]) were changed but insignificant. In addition, number of gram negative was not changed after placebo therapy (363 [291] vs. 298 [253]).

**Conclusion:** 7 days oral supplementation with cultured lactobacillus subtilis/streptococcus faecium was associated with restoration of bowel flora and improvement of the mDF and LPS in AH than placebo group.

Keywords: Hepatitis, Alcoholic, Probiotics, Cytokines, Lipopolysaccharides

**PS-2-6**

**Diagnostic Performance of Controlled Attenuation Parameter for Detection of Hepatic Steatosis in Patients with Chronic Liver Disease: A Prospective Study**

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**Background/Aim:** Controlled attenuation parameter (CAP) is a non-invasive method of measuring hepatic steatosis using a process based on transient elastography. We investigated the diagnostic accuracy of CAP in detecting hepatic steatosis in patients with chronic liver disease.

**Method:** A total 143 patients with chronic liver disease who underwent liver biopsy and CAP were consecutively enrolled in this prospective study. The performance of CAP for detection of hepatic steatosis compared with histological data was calculated using area under receiver operative characteristics curves (AUROC). Steatosis was categorized into S0 (66% of hepatocytes).

**Results:** Characteristics of the 143 patients included were as follow: age 69 years, BMI 24.6 kg/m2, viral liver disease 65%, NAFLD 18.9% and alcoholic liver disease 1.4%. Steatosis repar was: S0 69.2%, S1 21.0%, S2 7.0% and S3 2.8%. The mean CAP values were 234.5 dB/m for ≥S1, 241 dB/m for ≥S2 and 255 dB/m in S1; 272 dB/m in S2; 319 dB/m in S3, CAP was gradually increased along with steatosis grade (208 dB/m in S0; 255 dB/m in S1; 272 dB/m in S2; 319 dB/m in S3, P<0.001) and was significantly correlated with BMI, body weight, total cholesterol, triglyceride as well as steatosis grade. The AUROC of CAP were 0.818 for ≥S1 (0.842 for ≥S2 (P<0.001) and 0.923 for ≥S3 (P=0.004), respectively. For maximum sum of sensitivity and specificity, the optimal cutoff CAP values were 234.5 dB/m for ≥S1, 241 dB/m for ≥S2 and