Background/Aims: Alcohol consumption continues to be a common cause of acute and chronic liver disease. The study objectives were to present nationally representative findings on the prevalence and correlates of alcoholic liver disease (ALD).

Methods: Data from a representative sample of 7,893 adults in the Korean National Health and Nutrition Examination Survey 2009 were analyzed. ALD was defined as heavy alcohol consumption (≥40 gm/d for men or ≥20 gm/d for women) and elevated liver tests.

Results: Approximately 6.7% (95% confidence interval [CI], 6.0% to 7.4%) was at heavy alcohol consumption. Of these heavy drinkers, one-quarter also had ALD. The prevalence of ALD was 1.7% (95% CI, 1.3% to 2.1%). The prevalence was higher in men (2.7%, p<0.01) than in women (0.8%). There was no significant difference across age groups. Men had significantly higher odds of having ALD compared to women (odds ratio [OR]=3.3; 95% CI, 1.96 to 5.57). Current smoking (OR=7.96; 95% CI, 3.71 to 17.09), less than high school education (OR=2.65, 95% CI, 1.33 to 5.31), and lowest income (OR=3.51, 95% CI, 1.69 to 7.30) were also significantly associated with ALD.

Conclusions: These findings provide the most updated prevalence estimates of ALD in the general population and they highlight its strong association with smoking, gender differences, and lower socioeconomic status in the general population.

Declaration of Funding Source: This work was supported by 2010 KASL Research Fund. This work was supported by Grant from Inje University, 2010.

Keywords: Alcohol; Alcoholic liver disease; Prevalence

0-106
Outcome after emergency liver transplantation in adult patients with acute liver failure: trying to define when transplantation is futile
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Background: Not all patients undergoing emergency liver transplantation (LT) for acute liver failure (ALF) survive. Given the critical shortage of liver donors, futile as well as unnecessary LT should be avoided.

Methods: To investigate the predictive factors for post-LT mortality in patients with ALF, a database of 160 consecutive patients who underwent primary LT for ALF between 2000 and 2009 in our institution was analyzed. Glomerular filtration rate (GFR) was estimated according to the Modification of Diet in Renal Disease (MDRD) equation.

Results: Median age of 160 patients was 40 (range, 16-76). Male was 56.3%. Most common cause of ALF was hepatitis B virus infection (29.4%). Three quarter (n=124, 77.5%) of patients underwent adult-to-adult living-donor LT (LDLT) and one quarter (n=36, 22.5%) received deceased-donor LT (DDLT). Patients were followed-up for a median of 34 (range, 1-132) months after LT. The 1- and 3-year survival rate of patients were 78.1% and 74.6%, respectively. Ten patients received re-transplantation for graft failure, but 3 of them died within 4 month. There was no difference in patient and graft survival rates between LDLT and DDLT (P=0.1). Among variables at the time of LT, lower estimated GFR (HR 0.99, P=0.04), lower serum sodium (HR 0.95, P=0.03), use of inotropics (HR 5.89, P<0.01), and older donor age (HR 1.04, P=0.03) were independently associated with increased 1 year mortality after LT. None of the variables indicative of hepatic dysfunction, i.e., bilirubin or INR, was associated with post-LT outcome.

Conclusions: Survival after adult-to-adult LDLT is comparable to survival after DDLT in patients with ALF. Renal dysfunction, hyponatremia, use of inotropics, and older donor age but not hepatic dysfunction per se, at the time of LT were significant predictors of post-LT mortality in patients with ALF.

Keywords: Acute liver failure, Liver transplantation, Age, Glomerular filtration rate, Sodium

0-107
Monosodium urate attenuates bile acid induced hepatocyte apoptosis by modulating ER stress and JNK activation
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Background & Aims: Uric acid is constitutively present in cells and has a wide spectrum of actions, including being a pro-and anti-oxidant and a neurostimulant. Moreover, monosodium urate (MSU) released from damaged cells, named damage-associated molecular pattern molecules, acts as an activator of innate immune response, inflammation and induces protective mechanisms against necrotic cell death. However, there is little known about its effects on apoptotic cell death. This study investigated the effect of MSU against bile acid induced hepatocyte apoptosis.

Methods: Huh-BAT, SNU 761 and SNU 475 cells were used in this study. Cells were treated with MSU in the presence or absence of deoxycholate (DC). Cellular growth and apoptosis were analyzed by MTS assay and DAPI staining. Apoptotic signaling pathways were explored by immunoblot analysis.

Results: MSU treatment significantly inhibited DC-mediated hepatocyte apoptosis by attenuating DC-induced caspase 9 and 7 activation. However, MSU did not affect tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) induced apoptosis. MSU treatment diminished DC-induced c-Jun N-terminal kinase (JNK) activation and eukaryotic initiation factor 2a phosphorylation. In particular, MSU-mediated inhibition of DC-induced JNK activation and ER stress was found to be responsible for attenuating caspase activation.