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## Acceptance Notice for E-Poster Exhibition

Presented on March 31, 2022 at the 31<sup>st</sup> Annual Meeting of the Asian Pacific Association for the Study of the Liver (Abstract # PP-0581) held during March 30-April 03, 2022 at COEX Mall Convention Center, Gangnam-gu, Seoul, South Korea.

December 27, 2021

Dear Dr. Joseph George,

On behalf of the Organizing Committee, we are pleased to inform you that your abstract has been accepted for the **E-poster Exhibition** as follows:

Abstract Information	
Abstract Submission No.	PP-0581
Abstract Title	Combination treatment with curcumin and silibinin prevents oxidative stress and attenuates experimentally induced liver injury and fibrosis in rats
Presenting Author	Joseph George

***\*Your abstract will be presented in the E-poster exhibition zone as well as on the online platform during the conference period.***

***Please confirm your participation as a presenter by replying to us( [abstract.apasl2022seoul@gmail.com](mailto:abstract.apasl2022seoul@gmail.com) ) by no later than January 9, 2022. If there are changes to the presenter or if you plan to withdraw, you are requested to contact the secretariat before the deadline mentioned above.***

***We highly recommend that you to complete the early bird registration by December 31 (Fri), 2021 for APASL 2022 to benefit from the discounted registration fee.***

### Registration

To ensure your presence, the presenting author must complete his/her online registration with full payment. Abstracts will be published in the program book and abstract book. The presenting author should **register for APASL 2022 by the early bird registration deadline of December 31 (Fri), 2021 or the pre-registration deadline of February 20, (Sun), 2022.**

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**We understand that some of you may feel unsure of your participation at this time due to COVID-19 situation. Please note that you can get a full refund on your registration fee until February 28 (Mon), 2021, so please do not hesitate to register.**

Please note that abstracts may be withdrawn from being presented if the presenting authors of the accepted abstracts do not complete their registration on time.

**Details regarding the preparation and submission of your presentation file will be notified in January after you confirm your intent to present at the conference.**

We very much appreciate your continued interest in APASL 2022, and look forward to seeing you. We firmly believe that your contribution will make APASL 2022 all the more successful.

Should you have any inquiries, please do not hesitate to contact us([abstract.apasl2022seoul@gmail.com](mailto:abstract.apasl2022seoul@gmail.com))

Sincerely yours,



**Jin Mo Yang**

President, APASL 2022 Organizing Committee

**Results:** Of the outpatients, 17% (n = 153) aged  $54 \pm 12$  had LC and 97% patients had compensated LC without clinical manifestations. Five (5) patients had regular alcohol consumption (RAC). Most cases (93%)—chronic hepatitis C (CHC). All patients with chronic hepatitis B (CHB) had replication. Of the hospitalized patients, 633 (75%) age of  $50 \pm 14$  (29–74) had LC and 90%—subcompensated/decompensated. RAC was seen in 60%. CHC was accounted for 30%, CHB – for 34%, and out of these, 67% had occult hepatitis B (OHB), 22%—mixed, 14%—unknown etiology. All 455-deceased aged  $48 \pm 12$  (27–76) had LC. RAC was seen in 65%. The share of CHB was 23.7%, out of these 71%—OHB, CHC—35.2%, mixed—31.5%, 19.6%—unknown etiology. In the study of liver micropreparations from 97-deceased age  $45 \pm 12$ , only 77% of patients with subcompensated LC and 70%—with decompensated LC showed signs of completed cirrhotic transformation. In all cases of LC, the fact of multimorbidity was noted.

**Conclusion:** CHC dominates. There is insufficient screening OHB at the outpatient stage. Outpatient with LC are persons of working age, socially adapted and employed, without alcohol dependence, with compensated LC, with minimal clinical manifestations. Among inpatients and deceased socially maladapted individuals, and a high percentage of alcohol addicts are more common. Thirty percent (30%) of patients die from causes not related to decompensation of the underlying disease. This confirms the effect of comorbid pathology on CVH.

[OP-0569]

#### Frequency of sarcopenia in patients with cirrhosis and factors predicting sarcopenia

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**Objectives:** To determine frequency of sarcopenia in patients with cirrhosis and factors predicting sarcopenia.

**Materials and Methods:** A total of 386 patients with cirrhosis were enrolled. Sarcopenia was determined based on the Asian Working Group for Sarcopenia-2019. Patients' clinical and laboratory characteristics were recorded. Chi square test was applied to find out the association between sarcopenia and other demographic variables. Logistic regression analysis was performed to identify independent predictors for sarcopenia. P value of  $\leq 0.05$  was considered as significant.

**Results:** Out of 386 patients, sarcopenia was found in 314 patients (81.34%). Sarcopenia was more prevalent in females (86.8% vs 78.1%,  $p = 0.036$ ). Most common etiology was chronic viral hepatitis (85.5%). 33% had hepatoma, while 32% were on statins. Mean MELD-Na score was  $14.49 \pm 5.543$ . Mean age and BMI of study participants were 52.13 with SD of 8.46. and 23.8 with SD of 4.11 respectively. On multivariate analysis, female gender (odds ratio (OR): 1.813, 95%CI 1.033 – 3.182,  $P = 0.038$ ), Age > 49.5 years (OR: 2.925, 95%CI: 1.727 – 4.955,  $P < 0.001$ ), and albumin > 2.45 g/dl (OR: 0.28, 95%CI: 0.129 – 0.606) were associated with sarcopenia.

**Conclusion:** Sarcopenia is very common in patients with cirrhosis. Special measures should be taken for patients with risk factors of sarcopenia to improve their outcomes.

[OP-0576]

#### Bone mineral density loss in liver cirrhosis patients

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**Objectives:** One of the liver cirrhosis complications is related to bone metabolism is known as hepatic osteodystrophy (HO). Pathologic fracture caused by HO can significantly affect the quality of life and life expectancy of patients. The mechanism is multifactorial. Our study was carried out to assess the prevalence of HO and to identify the factors that related to the loss of bone mineral density in liver cirrhosis patients.

**Materials and Methods:** Consecutive eligible patients with liver cirrhosis were recruited. Bone mineral densitometry (BMD) was evaluated by dual-energy X-ray absorptiometry (DEXA). BMD at both femoral neck and lumbar spine were collected, and the lowest T score was used. Hepatic osteodystrophy was defined as a T score of  $\leq -1$ . Continuous variables were presented by mean or median as appropriate, and categorical variables by percentage. The univariate and multivariate analyses were performed to identify factors related to the development of hepatic osteodystrophy.

**Results:** Among 80 participants, 75% (n = 60) were men, 25% (n = 20) were women, and the median duration of liver disease was 3 years. The mean age and body mass index (BMI) was  $48.89 \pm 7.32$  years old;  $24.27 \pm 0.06$  respectively. Viral hepatitis was the most common etiology of cirrhosis (97.5%). There were 52.5% (n = 42) participants with Child-Turcotte-Pugh (CTP) score grade A, 30% (n = 24) with CTP score grade B, and 17.5% (n = 14) with CTP score grade C. The mean of lowest T score was  $-1.398 \pm 1.252$ . HO were present in 61.3% (n = 49) participants (41.3% osteopenia and 20% osteoporosis). On both univariate and multivariate analysis, BMI and duration of liver disease showed a significant association with HO ( $p = 0.003$ ;  $p = 0.001$ ).

**Conclusion:** Our study found a high prevalence (61.3%) of hepatic osteodystrophy in cirrhosis patients. Low BMI and a longer duration of liver disease were related to the development of HO in cirrhosis patients.

[PP-0581]

#### Combination treatment with curcumin and silibinin prevents oxidative stress and attenuates experimentally induced liver injury and fibrosis in rats

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**Objectives:** Hepatic fibrosis is characterized by excessive synthesis and deposition of connective tissue components, especially collagens in the extracellular matrix of the liver. Curcumin is a food additive with established antioxidant and anti-inflammatory properties. Silibinin is the active constituent of silymarin and a potent antioxidant

with proven pharmacological effects against alcoholic liver disease and non-alcoholic steatohepatitis. We studied the protective effects of curcumin and silibinin to prevent oxidative stress and attenuate experimentally induced hepatic fibrosis in rats.?

**Materials and Methods:** Liver injury and hepatic fibrosis was induced with intraperitoneal injections of N-nitrosodimethylamine (NDMA) in a dose of 10 mg/kg body weight daily for 10 consecutive days. Groups of animals received curcumin 200 mg/kg body weight and/or silibinin 20 mg/kg body weight everyday orally 2 h prior to the administration of NDMA and also until the end of the study. All the animals were sacrificed on day 21 from the beginning of exposure. Levels of AST, ALT, malondialdehyde, glutathione, ascorbic acid, and hyaluronan were measured either in serum, liver, or both. Immunohistochemistry was carried out for alpha-SMA, collagen type I and type III, and 4-hydroxy-2-nonenal (4-HNE). RT-PCR was performed for alpha-SMA, type I and type III collagens.

**Results:** Serial administrations of NDMA produced well developed fibrosis and early cirrhosis in rat liver. Combination treatment with curcumin and silibinin significantly reduced serum/hepatic levels of AST, ALT, malondialdehyde, glutathione, ascorbic acid and hyaluronan and completely prevented deposition of collagen fibers in the liver. Immunohistochemical staining and RT-PCR depicted marked decrease in the expression of alpha-SMA, 4-HNE, and collagens type I, and type III.

**Conclusion:** Combination treatment with curcumin and silibinin efficiently decreased oxidative stress, markedly reduced expression of collagens, and prevented hepatic fibrosis. The data demonstrated that curcumin and silibinin could be used as potent therapeutic agents to prevent liver injury and hepatic fibrosis.

[PP-0623]

**Salvianolic acid A is the active compound of *Salvia miltiorrhiza* against the activation of NLRP3 inflammasomes on macrophages**

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**Objectives:** *Salvia miltiorrhiza* (SM) is a Chinese herbal medicine that is widely used to treat liver diseases in clinic. We previously found SM was effective in treating liver injury via inhibiting expression of NLRP3 in vivo, but the active compound of SM and the underlying mechanism remained unclearly. Here, we aimed to screen its active substances against NLRP3 inflammasomes and explore the mechanism involved.

**Materials and Methods:** A cell culture model was investigated to detect NLRP3-dependent IL-1 $\beta$  release using bone marrow-derived macrophages (BMDMs). 16 ingredients of SM, including danshensu, magnesium lithospermate B, tanshinone I, tanshinone IIA, miltirone, manool, salvianolic acid A, salvianolic acid B, salvianolic acid C, dhydrotanshinone I, dihydroisotanshinone I, caffeic acid, neocryptotanshinone, cryptotanshinone, lithospermic acid, were screened out to confirm the maximum non-toxic concentration for BMDMs by LDH and CCK8 kits, respectively. For NLRP3 inflammasome activation, BMDMs were primed with lipopolysaccharide (LPS), and subsequently were stimulated with nigericin or adenosine triphosphate (ATP). The most active substances against NLRP3 inflammasomes in SM was screened out and the underlying mechanism were explored.

**Results:** Maximum non-toxic concentration and IC<sub>50</sub> of 16 ingredients for BMDMs were assayed. Among the ingredients,

Salvianolic acid A was the most anti-inflammatory substances in SM that played a dose dependent (25, 50 and 100  $\mu$ M) in alleviating the release of IL-1 $\beta$  on activated NLRP3 inflammasomes. Levels of mitochondrial ROS production and expression of LC3 and LC3-II in LPS (50 ng/ml)/ATP (2.5 mM)- and LPS (50 ng/ml)/nigericin (4  $\mu$ M)-stimulated BMDMs were significantly reduced respectively, which revealed that Salvianolic acid A might confer mitochondrial protection via promoting autophagy pathway.

**Conclusion:** Salvianolic acid A might be the efficacious compound in SM against activation of NLRP3 inflammasome on macrophages in vitro. The mechanism was associated with alleviating mitochondrial oxidative stress and promoting autophagy pathway in vitro.

[OP-0625]

**Neutrophillymphocyte ratio and the risk of 30-day mortality in patients with overt hepatic encephalopathy**

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**Objectives:** Overt hepatic encephalopathy (OHE) is related to a risk of adverse outcomes and short survival. However, the association between different neutrophil to lymphocyte ratio (NLR) levels and the 30-day mortality risk in cirrhotic patients with OHE has not yet been well assessed.

**Materials and Methods:** We retrospectively included 1301 patients with OHE at Beijing Ditan Hospital between August 2008 and December 2018. By adjusting for important risk variables, we investigated the association between NLR and the 30-day mortality risk using Cox regression analysis and restricted cubic splines. The discrimination and clinical usefulness of NLR were assessed by receiver operating characteristic curves and decision curve analysis.

**Results:** All patients were divided into four groups according to quartiles of the baseline NLR (< 2.5, 2.5 – 4.3, 4.3 – 7.5, > 7.5). The 30-day mortality were 7.8%, 12.7%, 19.5%, and 34.1%, respectively ( $p < 0.001$ ). Compared with the lowest quartile, increased NLR was correlated with increasing 30-day mortality after multivariable adjustment (NLR 2.5–4.3: adjusted hazard ratio [AHR], 1.17 (95% confidence interval [CI], 0.70–1.95); NLR 4.3 – 7.5: AHR, 1.58 (95% CI, 1.01 – 2.47); NLR > 7.5: AHR, 2.32 (95% CI, 1.50 – 3.57). A non-linear association between NLR and the adjusted probability of 30-day mortality was observed. There was a significant increasing 30-day mortality in the range of  $4.3 < \text{NLR} < 12$ . The performance of NLR + MELD (0.839) was higher than that of NLR + MELD-Na (0.829), MELD (0.814), and MELD-Na (0.812) in patients with OHE. The decision curve showed that NLR + MELD had superior standardized net benefit than MELD and NLR. Notably, elevated NLR was linked to increased incidence of 30-day mortality in the subgroup analysis of patients with OHE (HR > 1.0).

**Conclusion:** Elevated NLR is independently associated with 30-day mortality in cirrhotic patients with OHE.