

The 34th Annual Meeting of the Asian Pacific Association for the Study of the Liver

APASL 2025

BEIJING

Multidisciplinary Collaboration
For Elimination & Cure

26-30 March 2025
China National Convention Center
Beijing · China

ABSTRACT BOOK



Mortality associated with multidrug-resistance in Enterobacteriaceae infections in end-stage liver disease: a propensity score-weighted retrospective study

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Background: End-stage liver disease (ESLD) is a severe condition, contributing significantly to global mortality. Bacterial infections, especially from multidrug-resistant pathogen, are common in ESLD patients and worsen outcomes. There is insufficient data on the effect of Enterobacteriaceae resistance on the outcomes of these patients. This study aims to assess the impact of MDR Enterobacteriaceae infections on mortality rates in ESLD patients.

Method: This retrospective study included 257 ESLD patients with Enterobacteriaceae infections from 2018 to 2023, and separated into multidrug-resistant Enterobacteriaceae infections (MDR-EI) and non-multidrug-resistant Enterobacteriaceae infections (non-MDR-EI) group. Propensity score model with stabilized inverse probability of treatment weighting (IPTW-S) was conducted to balance covariates. The hazard ratio for 30-day mortality attributed to multidrug resistance was then estimated using Cox regression and Kaplan-Meier curves.

Result: MDR-EI caused 99 infections, of which 77 (77.8%) involved Extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae, and 15 (15.2%) carbapenem resistance. MDR-EI ESLD patients had a 30-day crude death rate of 25.3%, while non-MDR-EI patients had a rate of 8.2% ($p < 0.001$). After adjustment, ESLD patients with MDR-EI scored with a 30-day mortality hazard ratio of 2.273 (interquartile range: 1.109-4.660; $p = 0.010$). Plasma exchange treatment, urinary catheterization, previous antibiotic exposure, and used diuretics were independently associated with MDR-EI in ESLD.

Conclusion: Multidrug resistance significantly increases 30-day mortality in ESLD patients with Enterobacteriaceae infections. Effective preventive strategies for managing antibiotic resistance in this population should be urgently established.

Table and Figure: Figure 1. MDR-EI and non-MDR-EI crude mortality (A) and weighted mortality (B); HR, hazard ratio

Figure 2. Risk factors for (A) MDR Enterobacteriaceae infection in ESLD and the (B) 30-day crude mortality of enterobacteriaceae infections in ESLD

EP1056

Association between gallbladder polyps and hepatitis B virus infection

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Background: Hepatitis B virus (HBV) infection has been regarded as the common risk factor of gallbladder polyps (GP). This study aimed to investigate the potential association between GP and HBV replication among chronic HBV-infected individuals.

Method: We conducted a retrospective case-control study between 2019 and 2022, involving a total of 19,480 Chinese HBV-infected individuals. The GP cohort consisted of new-onset GP patients, while the control cohort matched based on gender, age and HBV-infected course using propensity score matching (PSM). We analyzed risk factors of GP by univariate and multivariate analyses. And we conducted subgroup analysis on primary exposures, including hepatitis B e antigen (HBeAg) expression and HBV DNA load.

Result: In this study, approximately 25.3% of HBV-infected individuals were diagnosed with GP through abdominal ultrasonography. Following PSM, 881 patients per cohort were included in the analyses. The results indicated that HBV replication, abnormal lipid and albumin metabolism were identified as risk factors for GP in HBV-infected individuals, while receiving antiviral therapy was found to be a protective factor. Patients with low-level viremia had a 58% higher risk of developing GP (adjusted odds ratio [aOR]=1.58, 95% confidence interval [CI]=1.10-2.28), compared to those with undetectable HBV DNA. Patients with positive hepatitis B e antigen (aOR=1.28, 95%CI=1.02-1.59) had increased risks of GP than those negative patients. Following six months of antiviral therapy, patients who failed to achieve virological response or HBeAg loss had higher risk of developing GP.

Conclusion: Low-level replication and HBeAg-positive were associated with higher risks of developing GP in chronic HBV-infected individuals, particularly in those who have received antiviral therapy for more than six months.

Table and Figure: Figure 1. The flow diagram of inclusion.

EP1058

Pemafibrate modulates peroxisome proliferator-activated receptor alpha and prevents alcohol-associated liver disease

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Background: Alcohol-associated liver disease (ALD) with steatohepatitis that could progress to liver cirrhosis is a major problem in chronic alcohol consumption. Pemafibrate is a novel, highly specific peroxisome proliferator-activated receptor- α (PPAR α) modulator that regulates the expression of target genes related to lipid and glucose metabolism. We evaluated the role of pemafibrate to modulate PPAR α and prevent steatosis and ALD in rats.

Method: The animals were treated with liquid diet containing ethanol (36% of total calories) or an isocaloric carbohydrate diet for 4 weeks. Subsequently, both groups were fed with either 0.5% aqueous methylcellulose solution (MC) or MC containing 0.3 mg pemafibrate/kg body weight orally twice a day along with the liquid diet for another 4 weeks. A set of animals were sacrificed at the 4th week before the start of pemafibrate treatment and the remaining animals at the end of 8 weeks. Blood and liver samples were collected for biochemical and histopathological evaluations.

Result: Treatment with pemafibrate prevented inflammation and steatosis in the hepatic tissue. Furthermore, pemafibrate administration markedly increased hepatic NAD and NADH levels, reduced both serum and hepatic triglyceride levels, and upregulated the expression of molecules involved in lipid metabolism.

Conclusion: The results of the present study demonstrated that pemafibrate modulates target genes related to hepatic lipid metabolism and prevents deposition of fat globules in the liver during chronic alcohol feeding in rats. Therefore, pemafibrate could be used as a potent therapeutic agent to prevent steatosis and related adverse events in ALD.

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EP1059

Youthful gut microbiome improves liver function and suppresses HCC in the elderly

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Background: Aging is a significant risk factor for cancer, contributing to the rising incidence of malignancies among older adults. Hepatocellular carcinoma (HCC), one of the fastest-growing cancers worldwide, is projected to affect 22 million individuals by 2032. Limited treatment options and challenges in early detection highlight the urgent need for safe, preventive strategies against HCC. This study