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refer and link pregnant women for treatment after pregnancy and breastfeeding; however, in practice, very few have completed successful treatment. To date, three case series have been published that include safety results for HCV treatment in pregnancy. ACOG recommends that DAAs be initiated only through a clinical trial and that pregnant women while taking a DAA should be counseled about the risks and benefits of continuing treatment.

To report the experience of the HAEV Hepatitis Clinic with the treatment of 3 pregnant women with HCV on DAAs during the second half of pregnancy with sustained viral response (SVR) and no adverse effects to the combination to date.

Patients and Methods: Since 2021, three cases of pregnant women with HCV infection confirmed by viral load have been presented. After evaluation and categorization as F0-F1 by FIB 4, they were treated with Sofosbuvir-Velpatasvir 400/100 mg for 90 days

Results: Patients were treated with Sofosbuvir-Velpatasvir 400/ 100 mg for 90 days, with no reports of perinatal abnormalities. The subsequent negative viral load was achieved in the pair. Only one patient reported headache and dizziness as adverse symptoms. After monitoring, a planned termination of pregnancy was decided to reduce the risk of vertical transmission, and counseling on proper breastfeeding techniques was provided to discontinue breastfeeding.

Conclusions: Sofosbuvir-Velpatasvir was administered for 12 weeks without adverse effects on the pair, and SVR was achieved at the time of treatment in the three treated patients, demonstrating the effectiveness and safety of the treatment. This provides a solution to a public health and maternal-fetal problem in our setting, which prevents perinatal transmission. Following these results, we propose evaluating its use in similar cases with the intention of contributing to the eradication of HCV infection.

Conflict of interest: None

https://doi.org/10.1016/j.aohep.2025.102002

#48

INR-PLATELET RATIO AS A PREDICTOR OF ESOPHAGEAL VARICES IN MEXICAN CIRRHOTIC PATIENTS

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Introduction and Objectives: High mortality from esophageal variceal bleeding necessitates primary prophylaxis in cirrhosis. Mexico's endoscopy-limited settings require biochemical predictors like the INR-Platelet Ratio (INPR) for variceal detection. The present work proposes that the INPR retains predictive validity for esophageal varices in Mexican cirrhotic patients. Consequently, validation of this hypothesis constitutes the primary objective of this investigation.

Patients and Methods: An observational, single-center study was conducted in the Gastroenterology Department of Hospital Juárez de México between 2023 and 2024.Inclusion criteria:Patients aged over 18 years with a diagnosis of cirrhosis confirmed by FIB-4 or hepatic ultrasound, and no prior endoscopic screening. A total of 139 patients were included: 71 women (51.1%) and 68 men (48.9%). Statistical analyses were performed using IBM SPSS Statistics software. Descriptive population analyses utilized frequencies and medians. Group

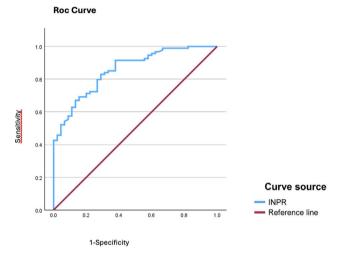
comparisons were conducted using the chi-square test and Student's t-test, with a p-value <0.05 considered statistically significant.ROC curves and the Youden index were employed to identify optimal cut-off values for sensitivity and specificity.

Results: Using an INPR cut-off of ≥0.9463 for detecting esophageal varices (irrespective of size), the following performance metrics were achieved: sensitivity 83%, specificity 71%, PPV 85%, NPV 66%. +LR 2.87, -LR 0.24.

Conclusions: The INR-Platelet Ratio is an efficient tool for health-care providers to initiate screening and prioritize early endoscopy, particularly in patients with other risk markers such as thrombocytopenia or Child-Pugh B/C cirrhosis. Future studies should evaluate its cutoff points to reduce unnecessary endoscopies and improve timely complication detection.

Conflict of interest: None

ROC curve for esophageal varices



https://doi.org/10.1016/j.aohep.2025.102003

#50

LONGITUDINAL CHANGES IN STEATOTIC LIVER DISEASE SUBTYPE CLASSIFICATION AND SUBSEQUENT RISK OF MAJOR ADVERSE LIVER OUTCOMES

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Introduction and Objectives: Steatotic liver disease (SLD) includes metabolic dysfunction-associated steatotic liver disease (MASLD), alcohol-associated liver disease (ALD), and their intersection (MetALD). SLD subtype classification may change over time; however, the impact of these transitions on major adverse liver outcomes (MALO) is unknown.

Materials and Methods: We conducted a retrospective study of adults with imaging-confirmed steatosis (n=270,302) in the Veterans Health Administration (2010-2021). The primary exposure was change in SLD subtype classification between cohort entry (steatosis on imaging) and a 2-year landmark. The primary outcome was incident MALO (cirrhosis, decompensation, HCC, transplant, liver-related death). We calculated incidence rates per 100 person-years and multivariable cause-specific Cox regression models to examine the magnitud of the association between changes in SLD subtype and subsequent MALO.

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Results: At the 2-year landmark, 8.2% of those with baseline MASLD were reclassified to MetALD or ALD, 34.2% of those with baseline MetALD were reclassified to MASLD or ALD, and 64.0% of those with baseline ALD were reclassified to MASLD or MetALD. Among baseline MASLD, the risk of MALO was higher for those reclassified to MetALD (HR 1.55;95% CI 1.40-1.71) or ALD (HR 2.13;95% CI 1.66-2.74) compared with those who remained MASLD. Among baseline MetALD, the risk of MALO was lower for those reclassified to MASLD (HR 0.55;95% CI 0.48-0.64) and higher for those reclassified to ALD (HR 1.80;95% CI 1.58-2.06) compared with those who remained Met-ALD. Among baseline ALD, the risk of MALO was lower for those reclassified to MASLD (HR 0.31;95% CI 0.21-0.46) or MetALD (HR 0.82;95% CI 0.70-0.96) compared with those who remained ALD.

Conclusions: Changes in SLD subtype classification are associated with distinct MALO risks.

Conflict of interest: Yes, NIH T32 DK007740

Association of changes in steatotic liver disease subtype classification with major adverse liver outcomes

Baseline	2-Year Landmark	No. (%)	Events	Person-Years	IR per 100 PY (95% CI)	Hazard Ratio (95% CI) ^a
MetALD	15,720 (7.4)	431	63,561	0.68 (0.62-0.75)	1.55 (1.40-1.71)	
ALD	1,561 (0.7)	62	6,515	0.95 (0.74-1.22)	2.13 (1.66-2.74)	
MetALD	MetALD	30,623 (65.8)	1,125	127,571	0.88 (0.83-0.93)	1 [Reference]
	MASLD	11,686 (25.1)	229	48,907	0.47 (0.41-0.53)	0.55 (0.48-0.64)
	ALD	4,236 (9.1)	279	16,976	1.64 (1.46-1.85)	1.80 (1.58-2.06)
ALD	ALD	4,260 (36.0)	272	16,639	1.63 (1.45-1.84)	1 [Reference]
	MASLD	1,325 (11.2)	28	5,692	0.49 (0.34-0.71)	0.31 (0.21-0.46)
	MetALD	6.256 (52.8)	335	25.391	1.32 (1.19-1.47)	0.82 (0.70-0.96)

MetALD (,256 (52.8) 335 25,391 1.32
hol-associated liver disease; CI, confidence interval; IR, incidence rate; MASLD, metabolic dysfunction and alcohol-associated liver disease; No, number; PY, person-year

Note: Percentages have been rounded and may not total 100.

* Major adverse liver outcome was defined as the first occurrence of cirrhosis, decompensation, hepatocellular carcinoma, liver transplant, and liver-related death. Hazard ratios were estimated using cause-specific Cox proportional hazards regression models, adjusted for age, sex, smoli ing status, social deprivation index, and index year.

https://doi.org/10.1016/j.aohep.2025.102004

#51

ATORVASTATIN AND GENE EXPRESSION SIGNATURES IN HEPATOCARCINOGENESIS

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Introduction and Objectives: Hepatocellular carcinoma (HCC) represents a significant global health burden as the fourth leading cause of cancer-related deaths. While statins have shown promise in HCC prevention, their molecular mechanisms remain poorly understood.

We investigated the effect of atorvastatin (AT) on gene expression profiles and hepatocarcinogenesis in a hexachlorobenzene (HCB)induced HCC model.

Materials and Methods: Male Wistar rats were divided into four groups: control, AT (5 mg/kg), HCB (100 mg/kg), and AT+HCB. After 30 days of treatment, we analyzed hepatosomatic index, liver histology, and performed RNA sequencing to evaluate transcriptomic changes. Gene Set Enrichment Analysis and KEGG pathway analysis were used to identify key molecular pathways. Protein expression of selected targets was confirmed by immunohistochemistry.

Results: HCB treatment significantly increased hepatosomatic index (28%, p<0.01) and induced preneoplastic lesions, which were prevented by AT co-administration. RNA sequencing revealed HCB activated multiple oncogenic pathways, including RHO GTPase cycle, TGF- β , and receptor tyrosine kinase signaling, with 84.8% concordance with established cancer pathway genes. AT treatment upregulated protective PPAR signaling, autophagy, and cellular stress response pathways while downregulating oncogenic pathways activated by HCB. AT significantly reduced the expression of key oncogenic proteins including TGF- β 1, p53, and c-Myc in HCB-treated liver tissue.

Conclusions: Atorvastatin effectively prevents HCB-induced hepatocarcinogenesis through multiple mechanisms, including modulation of key oncogenic pathways and promotion of protective cellular responses. These findings provide new insights into the molecular mechanisms of statin-mediated HCC prevention and identify potential therapeutic targets for future interventions.

Conflict of interest: Yes, National Council of Scientific and Technological Research (PIP GI-11220200100397CO), University of Buenos Aires (PID 20020170100278BA) and intramural Funds to the Clinical Cooperation Unit Healthy Metabolism, Medical Faculty Mannheim, Heidelberg University.

https://doi.org/10.1016/j.aohep.2025.102005

#55

REPRIORITIZING THE LIVER TRANSPLANT WAITING LIST: IMPACT OF AUTOMATIC MELD-NA 29 FOR REFRACTORY ASCITES

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Introduction and Objectives: In Brazil, liver allocation follows the MELD-Na score. To address high-risk conditions not reflected by high scores, special situations are evaluated individually. Refractory ascites and hepatocellular carcinoma (HCC) account for \sim 80% of such cases. In 2021, Technical Note No. 32/2021 granted 29 MELD-Na points to patients with refractory ascites to improve access to liver transplant (LT).

Primary, to compare the time from special situation approval to LT in cases of refractory ascites and HCC, before and after the new policy. Secondary, assess transplant volume and waiting list mortality in both groups.

Patients and Methods: Retrospective, single-center study including adult patients granted special situation for refractory ascites or HCC in 2018-2020 (pre-policy) and 2022-2024 (post-policy). Cases from 2021 were excluded. Outcomes were time to LT, mortality on the waiting list, and transplant numbers.

Results: In refractory ascites, median time to LT decreased by 95 days (186 to 91; -51.1%). In HCC, waiting time increased by 36 days (197 to 233; +18.3%). Waiting list mortality dropped in both groups: from 16% to 7% for refractory ascites and from 11% to 6% for HCC. The absolute number of transplants remained stable across periods.

Conclusions: Technical Note 32/2021 had a direct positive impact on patients with refractory ascites. However, it was associated with increased waiting time for HCC patients, although without increased mortality. These findings highlight the need for continuous monitoring of allocation policies and broader multicenter evaluation.

Conflict of interest: None

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