

Background and aims: The EASL 2025 guideline introduced a simplified algorithm for managing chronic hepatitis B (CHB), recommending earlier initiation of antiviral therapy (AVT) based on viral load and fibrosis stage. We evaluated whether AVT initiation according to the EASL 2025 guideline is associated with a reduced risk of hepatocellular carcinoma (HCC).

Method: This multinational cohort study included 24,788 treatment-naïve CHB patients from 7 centers in South Korea, Hong Kong, and Taiwan (2007–2022). Predicted HCC risk assuming AVT initiation was estimated using PAGE-B (for groups including patients with cirrhosis) or PAGED-B scores (for groups including patients without cirrhosis). Patients were categorized according to the EASL 2025 guideline as follows: Groups 1 and 2 included patients with advanced fibrosis or cirrhosis without (Group 1) or with (Group 2) detectable HBV DNA; Group 3, HBV DNA $\geq 2,000$ IU/ml with significant fibrosis or alanine aminotransferase (ALT) $>$ upper limit of normal (ULN); Group 4, HBV DNA $\geq 2,000$ IU/ml without significant fibrosis or ALT $>$ ULN; and Group 5, HBV DNA $< 2,000$ IU/ml with ALT $\leq$ ULN. For each group, observed 5-year HCC incidence in untreated patients was compared with the predicted incidence derived from the models, assuming AVT initiation. An additional AVT-treated cohort ($n = 8,414$) was propensity score-matched to the untreated cohort to compare HCC risk between treated and untreated patients in Groups 2 and 3.

Results: When fibrosis was assessed by transient elastography, observed HCC risk in Group 1 was comparable to that predicted by PAGE-B. In contrast, observed HCC risk in untreated patients was significantly higher than predicted by PAGE-B in Group 2 (standardized incidence ratio [SIR] = 2.539, 95% confidence interval [CI] = 2.278–2.818) and by PAGED-B in Group 3 (SIR = 3.116, 95% CI = 1.755–5.045) (all $p < 0.001$). In Groups 4 and 5, observed and predicted HCC risks were comparable (all $p > 0.05$). These findings were consistent when fibrosis was assessed using fibrosis-4 or aspartate aminotransferase-to-platelet ratio index. In propensity score-matched analyses incorporating an additional cohort of patients treated with AVT, AVT initiation was associated with a significantly lower risk of HCC compared with no treatment in both Group 2 and Group 3 ($p = 0.035$ and $p = 0.007$, respectively).

Conclusion: AVT initiation based on viral load and fibrosis stage according to the EASL 2025 guideline is associated with a reduced HCC risk.

SAT-575

Epidemiology of hepatitis delta virus in Belgium: interim results of a prospective cross-sectional cohort study

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Background and aims: With new treatment options for chronic hepatitis delta virus (HDV) infection emerging, it is crucial to optimize screening and linkage to care. This prospective, cross-sectional study aims to systematically assess the HDV seroprevalence, viremia, and circulating genotypes (gt) in hepatitis B surface antigen (HBsAg) positive patients in Belgium. Also, the sensitivity of HDV antibody (HDVAb) testing in immunocompromised (IC) patients and patients suspected of having an acute HBV-HDV co-infection, will be evaluated.

Method: From 15 large Belgian hospitals, all consecutive samples testing positive for HBsAg during the study period (2024/11/25–2025/10/27), were sent to Sciensano (National Reference Center for Viral Hepatitis). Double HDV reflex testing was performed (HDVAb total: ELISA assay DiaSorin; HDV PCR: Realstar[®] kit Altona Diagnostics; in house HDV genotyping). Irrespective of the HDVAb result, HDV RNA screening was performed for all IC patients and those suspected of having an acute co-infection. Advanced fibrosis was defined based on elastography (HBV > 8 kPa; HDV > 10.4 kPa), histology and clinical cirrhosis criteria.

Results: HDV seroprevalence is 6.14% ($n = 129/2101$; 5.19%–7.25%). Of 129 HDVAb+ patients, 49 tested HDV RNA+. Of all IC patients ($n = 91$) and those with presumed acute co-infection ($n = 18$), 1 co-infected HBV-HIV+ patient tested HDV RNA+ (5.46 IU/mL) despite negative HDVAb. Therefore, HDV viremia in the entire cohort is 2.38% ($n = 50/2100$; 1.81%–3.13%), with predominant gt 1 ($n = 34$) and gt 5 ($n = 8$). HDVAb+ patients were more often female (51.2% vs 48.8%, $p = 0.022$) and were more likely to be HCV seropositive (7.0% vs 1.9%, $p = 0.010$). HDV RNA+ patients had higher FIB-4 (1.67 vs 1.13, $p < 0.001$), GGT (gamma-glutamyl transferase, 47 vs 23 U/L, $p < 0.001$), bilirubin (0.63 vs 0.50 mg/dL, $p = 0.010$), and INR values (international normalized ratio, 1.10 vs 1.00, $p < 0.001$). In a subset of 304 HBsAg+ patients [262 HDVAb-, 26 HDVAb+/RNA-, and 16 HDVAb+/RNA+ patients (2 newly

POSTER PRESENTATIONS

diagnosed)] from 5 hospitals, HDVAb+ patients more often had a history of injecting drug use (10.5% vs 0%, $p = 0.034$) and HDV viremia was strongly associated with advanced fibrosis (adjusted OR 22.2, CI 5.00–98.24, $p < 0.001$), independently of BMI and alcohol consumption.

Conclusion: Active HDV co-infection presents with more advanced liver disease and is expected in around one in every 40 HBsAg+ patients in Belgium. Wider implementation of HDV reflex-testing in Belgium is needed.

SAT-576

Hepatitis B virus genotype distribution and viral load patterns in African immigrants to Canada

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Background and aims: Hepatitis B virus (HBV) genotypes have distinct geographic and demographic distributions with important clinical implications. In Canada, immigration-driven HBV epidemiology provides globally diverse genotype and sociodemographic patterns reflecting regions of origin including Africa.

To characterize HBV genotype distribution and examine associations between genotype, sociodemographic factors, and HBV DNA levels in African immigrants to Canada.

Method: We conducted a retrospective cohort study of patients with HBV genotype results (2001–2025) in The Ottawa Hospital Viral Hepatitis Program. Genotype data were linked to sociodemographic characteristics. Associations between genotype and viral load were examined among treatment-naïve patients.

Results: Among 778 patients (median age 42.5 years; 52% male), 94% were born outside Canada, representing 78 countries and predominantly Asian (51%) and Black (35%) populations. Six genotypes were identified: A (19.8%), B (25.6%), C (21.0%), D (17.4%), E (15.9%), and F (0.4%). Distribution reflected global migration patterns: A in East Africa and the Caribbean, E in West Africa, B/C in East-Southeast Asia, and D across Middle East and North Africa (MENA), South Asia, and Europe/Central Asia. Genotype-race patterns were concordant (A/E predominantly Black; B/C >95% Asian; D heterogeneous). Age at presentation differed by genotype and race ($p < 0.0001$), with African-associated genotypes (A/D/E) and Black and Arab/MENA patients presenting younger than those with B/C. HBV viral load differed by genotype (A–E, $p < 0.0001$), with lower median log₁₀ HBV DNA in A, D and E (2.9–3.0 IU/mL) than in B and C (3.6 and 3.3 IU/mL). In multivariable models, genotype (particularly B/C), male sex and younger age were associated with higher baseline HBV DNA. Genotype was associated with antiviral initiation ($p < 0.0001$), lowest in A/E (26–28%) and highest in B/C (42–50%).

Conclusion: In this globally diverse, immigration-driven cohort, African-associated genotypes (A, D, E) and Black patients presented at younger ages, providing an opportunity to apply international recommendations for earlier HCC surveillance in high-risk African-origin populations. Among other factors, lower HBV DNA levels may inaccurately dissuade antiviral initiation in this at risk population.

SAT-577

HLA genetic markers identify HDV patients at risk of persistent viremia and advanced fibrosis

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Background and aims: Hepatitis delta virus (HDV) infection shows highly variable outcomes, from spontaneous viral control to rapid fibrosis progression. Identifying host factors that predict disease course is critical for early risk stratification and personalized management. Human leukocyte antigen (HLA) molecules orchestrate antiviral immunity, yet their clinical impact in HDV infection remains unclear. Here, we investigated HLA genetic markers associated with virological control and fibrosis progression.

Method: Blood samples from 75 anti-HDV-positive patients underwent high-resolution genotyping of classical HLA class I (A, B, C), class II (DPB1, DQB1, DRB1), and non-classical (HLA-G, HLA-E) genes. Patients were stratified by HDV RNA status (controllers: undetectable HDV-RNA in ≥ 2 consecutive samples, $n = 38$; viremic: persistently detectable HDV-RNA, $n = 37$) and fibrosis stage (METAVIR F0–F2, $n = 38$; F3–F4, $n = 37$). Associations were assessed using univariate and multivariable Firth's penalized logistic regression models. One hundred fifty healthy donors served as controls.

Results: HLA-A*11:01 was significantly enriched among viremic patients (27% vs 2.6% of controllers; OR = 12.9, 95% CI 1.8–569.4; $p = 0.002$) and was strongly linked to persistent viremia in multivariate analysis ($p = 0.02$). Advanced fibrosis was associated with HLA-A*11:01 and HLA-DPB1*17:01 (>21% of patients), while HLA-DPB1*01:01 predominated in mild fibrosis (34.2%). While all alleles were associated with fibrosis stage in univariate analyses ($p < 0.05$), only HLA-DPB1*17:01 remained independently associated with advanced fibrosis in multivariate analysis ($p = 0.03$). In addition, the presence of HLA-A*11:01 emerged as a factor independently associated with both the presence of detectable HDV-RNA and advanced fibrosis (8/25 patients; $p = 0.03$).

Conclusion: Specific HLA alleles may help to identify HDV patients at high risk of persistent viremia and progressive fibrosis. Incorporating HLA genotyping could enable earlier risk stratification and guide personalized monitoring and therapeutic decisions, offering a precision medicine approach in HDV management.

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SAT-578

A systematic literature review of quantitative HBsAg distribution, HBsAg/HBeAg loss and clinical outcomes in HIV/HBV co-infected population

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Background and aims: About 1% (~2.7 million) of patients (pts) with chronic hepatitis B virus (HBV) infection are coinfecting with human immunodeficiency virus (HIV), and 8–10% with HIV have chronic HBV. Coinfection accelerates HBV-related liver disease. HBV-active antiretroviral therapy (ART) suppresses viral replication but rarely achieves hepatitis B surface antigen (HBsAg) loss. This systematic literature review (SLR) assessed epidemiology, treatment patterns, quantitative HBsAg (qHBsAg) and clinical outcomes in HIV/HBV coinfection.

Method: We searched Embase and MEDLINE (01/01/2015–24/06/2025) for HIV/HBV coinfection studies (SLRs, narrative reviews, observational studies and clinical trials [for HBsAg data]; excluding preclinical and paediatric studies [< 18 years], case reports and small