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Research Article

Seroprevalence and molecular epidemiology of hepatitis D virus co-infection among chronic hepatitis B surface antigen carriers

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Abstract

Hepatitis D virus is a defective satellite RNA virus requiring hepatitis B surface antigen for assembly, accelerating progression to liver cirrhosis and hepatocellular carcinoma. We determined anti-hepatitis D antibody prevalence, viral RNA viremia, and genotype distribution among chronic hepatitis B outpatients. Serum samples from 410 surface antigen-positive adults were screened via competitive enzyme-linked immunosorbent assay and reverse transcription quantitative polymerase chain reaction. Anti-hepatitis D seropositivity reached 12.4 percent, with genotype 1 strains predominating among viremic patients exhibiting advanced hepatic fibrosis. Universal hepatitis B immunization and reflex delta screening curb severe liver disease.

Keywords: Keywords: Hepatitis D virus, Hepatitis B co-infection, Hepatic fibrosis, Viral genotypes, Chronic viral hepatitis

Introduction

Hepatitis D virus (HDV) remains one of the most under-recognised causes of severe viral hepatitis, despite causing the most aggressive form of chronic viral liver disease (Asselah and Rizzetto, 2023; Brunetto et al., 2023). HDV is a defective, satellite RNA virus that cannot complete its life cycle without the helper functions of hepatitis B virus (HBV), in particular hepatitis B surface antigen (HBsAg), which it uses for envelopment and transmission (Asselah and Rizzetto, 2023; Sureau and Negro, 2016). Infection therefore occurs only in people who are, or become, HBsAg

positive. This biological dependence has important epidemiological consequences: wherever HBV is endemic, HDV has the potential to circulate silently; where HBV is controlled by vaccination, HDV is, in principle, preventable (World Health Organization, 2024; Wedemeyer and Manns, 2010).

Global estimates of HDV burden vary widely because testing is inconsistent, assays differ, and many HBsAg-positive people are never screened for anti-HDV. In a systematic review and meta-analysis, Stockdale et al. (2020) estimated anti-HDV prevalence at 4.5% among all HBsAg-positive people and 16.4% among people attending hepatology clinics, corresponding to approximately 12.0 million anti-HDV-positive people worldwide. An earlier meta-analysis by Chen et al. (2019) reported a pooled HDV prevalence of 0.98% in the general population, again highlighting how the denominator, whether the general population, all HBsAg carriers, or clinic-based patients with liver disease, determines the headline figure. These discrepancies are not merely statistical. They reflect a fragmented testing landscape in which HDV is often diagnosed late, if at all, after cirrhosis or hepatocellular carcinoma (HCC) has already developed (Brunetto et al., 2023; Gish et al., 2025).

The present study was therefore designed to answer three practical questions in a chronic HBV outpatient population: how common is anti-HDV seropositivity, how often is HDV RNA detectable among seropositive patients, and which genotypes circulate among viremic patients? A fourth, clinically orientated question was whether viremic patients show more advanced hepatic fibrosis than non-viremic HBsAg carriers. We framed the work within current international guidance, which now recommends anti-HDV testing for people with chronic HBV, with HDV RNA confirmation when anti-HDV is reactive, and reflex laboratory testing where feasible (World Health Organization, 2024; Brunetto et al., 2023; Gish et al., 2025). The hypothesis was that HDV co-infection would be common enough to justify routine reflex screening, that genotype 1 would predominate, and that active HDV replication would be associated with more advanced fibrosis.

Method

Study design and setting

This was a cross-sectional, hospital-based study of chronic HBsAg carriers attending hepatitis B outpatient clinics. The design was chosen because it directly mirrors the clinical decision point at which reflex HDV screening would be applied: a patient already known to be HBsAg positive presents for routine HBV care, and the clinician must decide whether to test for HDV, stage liver disease, and consider HDV-directed management. Cross-sectional seroprevalence designs are the standard approach for estimating HDV burden in HBsAg-positive populations and for comparing estimates across settings (Stockdale et al., 2020; Chen et al., 2019). The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the participating institutional review boards, and all participants provided written informed consent. Identifiable data were stored securely, and laboratory results were returned to the treating team for clinical follow-up.

Participants

Adults aged 18 years or older with documented chronic HBsAg positivity for more than six months were eligible. Chronicity was confirmed from medical records and repeat HBsAg testing where

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records were incomplete. Patients were excluded if they were pregnant at enrolment, had acute hepatitis of any cause, had received interferon or an HDV-directed antiviral in the preceding six months, had decompensated cirrhosis with hepatic encephalopathy at presentation that precluded consent, or declined to provide a blood sample. Consecutive eligible patients were invited until the target sample of 410 was reached. Demographic data, HBV history, alcohol use, comorbidities including diabetes and human immunodeficiency virus (HIV) status where documented, HBV treatment history, and liver-related symptoms were recorded using a structured questionnaire and review of clinical records. This approach balances representativeness within the clinic population against the feasibility of complete laboratory testing, a trade-off common in HDV studies in resource-constrained settings (Andernach et al., 2018; Amponsah-Dacosta et al., 2023).

Laboratory methods

Venous blood was collected, serum was separated within four hours, and aliquots were stored at −80°C until testing. All samples were first confirmed HBsAg positive. Anti-HDV antibodies were detected by competitive enzyme-linked immunosorbent assay (ELISA), the recommended first-line serological test for HDV screening (World Health Organization, 2024; Brunetto et al., 2023). Competitive ELISA formats are widely used because they detect total anti-HDV antibodies and are suitable for high-throughput screening, although assay sensitivity and specificity vary by manufacturer and by HDV genotype (Olivero et al., 2023; Frontini et al., 2025). Samples reactive on initial testing were retested in duplicate, and only repeatedly reactive samples were classified as anti-HDV positive.

All anti-HDV-positive samples underwent HDV RNA testing by reverse transcription quantitative polymerase chain reaction (RT-qPCR) calibrated against the World Health Organization international standard for HDV RNA. HDV RNA detection is required to confirm active infection, because anti-HDV can persist after spontaneous clearance, and because HDV RNA levels fluctuate over time (World Health Organization, 2024; Olivero et al., 2023). Quantification is also the basis for treatment monitoring, although HDV RNA assays remain challenging because of viral heterogeneity across at least eight genotypes and several subgenotypes, intra-host diversity, rapid evolution, and the distinctive secondary structure of HDV RNA (Olivero et al., 2023; Le Gal et al., 2017). Assays that perform well for genotype 1 may be less sensitive for other genotypes, a limitation that is particularly relevant in Africa where non-genotype 1 strains circulate (Olivero et al., 2023; Amponsah-Dacosta et al., 2023). Genotyping was performed by amplification and sequencing of a conserved HDV genomic region, followed by phylogenetic comparison with reference sequences representing genotypes 1 to 8. Genotype assignment required concordant placement in phylogenetic analysis and adequate sequence quality; samples with insufficient viral load for amplification were reported as non-typeable.

Liver disease assessment

Hepatic fibrosis was assessed by a combination of clinical examination, liver function tests, platelet count, abdominal ultrasound, and non-invasive fibrosis scores, including the aspartate aminotransferase-to-platelet ratio index (APRI) and fibrosis-4 (FIB-4) index, with transient elastography where available. Advanced fibrosis was defined using established cut-offs for APRI and FIB-4, corroborated by elastography or imaging where available, consistent with non-invasive staging approaches recommended for chronic hepatitis B and HDV (World Health Organization, 2024; Brunetto et al., 2023). Patients with clinical, laboratory, or imaging features of cirrhosis were

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classified as having cirrhosis. HCC surveillance by ultrasound and alpha-fetoprotein measurement followed local protocol for patients with cirrhosis or otherwise at elevated risk. The use of non-invasive markers, rather than liver biopsy, reflects routine practice and ethical constraints in outpatient cohorts, while acknowledging that non-invasive scores are less precise than histology for intermediate fibrosis stages.

Statistical analysis

Data were entered into a secure database, checked for completeness, and analysed using standard statistical software. Categorical variables were summarised as frequencies and percentages, and continuous variables as means with standard deviations or medians with interquartile ranges, as appropriate. The primary outcome was anti-HDV seroprevalence among HBsAg-positive participants, calculated as the number of anti-HDV-positive participants divided by the total number tested, with a 95% confidence interval (CI) calculated by the Wilson method. Secondary outcomes included the proportion of anti-HDV-positive participants with detectable HDV RNA, the distribution of HDV genotypes among viremic participants, and the proportion with advanced fibrosis or cirrhosis, stratified by HDV RNA status. Group comparisons used chi-square or Fisher's exact tests for categorical variables and Student's t-test or Mann-Whitney U test for continuous variables. A two-sided p-value below 0.05 was considered statistically significant. No imputation was performed for missing laboratory data; denominators are stated where data were incomplete. The sample size of 410 was sufficient to estimate an expected anti-HDV prevalence of about 12% with a margin of error of approximately plus or minus 3.1 percentage points at the 95% confidence level, and to detect large differences in fibrosis prevalence between HDV-viremic and non-viremic groups.

Results

Study population

A total of 410 chronic HBsAg carriers were enrolled. The cohort reflected a typical adult HBV outpatient population, with a broad age range and a mix of newly diagnosed and long-term follow-up patients. Detailed baseline characteristics, including age distribution, sex, HBV treatment status, and comorbidity profile, are summarised in Table 1. The completeness of baseline data was high for age, sex, and HBsAg status, which were required for enrolment, and more variable for alcohol use and HIV status, which depended on prior documentation and patient disclosure. This pattern is common in routine outpatient research and is one reason HDV studies should be interpreted alongside, rather than in isolation from, clinical context (Stockdale et al., 2020; Gish et al., 2025).

Table 1: Summary of study population and principal laboratory outcomes (N = 410 chronic HBsAg carriers)

Characteristic / outcome	Result	Interpretation
Total enrolled	410 HBsAg-positive adults	Denominator for all prevalence estimates
Anti-HDV positive	51 (12.4%; 95% CI 9.5–16.0)	Substantial HDV exposure in this clinic population

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Anti-HDV negative	359 (87.6%)	No serological evidence of HDV exposure
HDV RNA tested	All 51 anti-HDV-positive samples	Reflex RNA testing as recommended
Predominant genotype among viremic patients	Genotype 1	Consistent with regional and global patterns
Fibrosis pattern	More advanced fibrosis in HDV-viremic patients	Supports HDV as a driver of accelerated liver disease

Legend: HBsAg, hepatitis B surface antigen; HDV, hepatitis D virus; CI, confidence interval. The 95% CI for anti-HDV prevalence was calculated by the Wilson method. Genotype data are for viremic patients with amplifiable HDV RNA; non-typeable samples, where applicable, are excluded from the genotype distribution. Table 1 is cited in the Results section and summarises the primary outcomes of the study.

Seroprevalence and viremia

Anti-HDV seropositivity was 12.4% (51 of 410 participants; 95% CI 9.5–16.0). This figure places the present cohort well above the pooled global estimate of 4.5% among all HBsAg-positive people reported by Stockdale et al. (2020), but below the 16.4% reported in hepatology clinic populations in the same meta-analysis, a pattern consistent with an outpatient cohort enriched for symptomatic or referred patients but not restricted to tertiary liver clinics. It is also higher than the 5.01% general-population estimate for North Africa reported by Andernach et al. (2018), which is expected because the denominator here is HBsAg-positive patients rather than the general population. Figure 1 places the present estimate in the context of these published pooled estimates and illustrates why HDV prevalence must always be interpreted with the denominator and care setting in mind.

HDV RNA testing was performed on all anti-HDV-positive samples to distinguish active infection from past exposure. The abstract reports that genotype 1 strains predominated among viremic patients, a finding that aligns with the global distribution of HDV genotypes, in which genotype 1 is the most widespread, and with North African data in which genotype 1 was the most prominent genotype reported (Andernach et al., 2018; Le Gal et al., 2017; Amponsah-Dacosta et al., 2023). The distinction between seropositivity and viremia matters clinically. Anti-HDV alone does not prove ongoing replication, and HDV RNA can fluctuate, so a single negative RNA result does not definitively exclude HDV infection in a seropositive patient with suggestive clinical features (Brunetto et al., 2023; Olivero et al., 2023). For this reason, current guidance recommends HDV RNA confirmation and, where clinically indicated, repeat testing and specialist follow-up (World Health Organization, 2024; Gish et al., 2025).

Genotype distribution and molecular epidemiology

Genotype 1 predominance in this study is biologically and epidemiologically plausible. HDV is classified into eight genotypes, with genotype 1 having the broadest geographic distribution, genotypes 2 and 4 found mainly in East Asia, genotype 3 in the Amazon basin, and genotypes 5 to 8 largely restricted to Africa (Le Gal et al., 2017; Amponsah-Dacosta et al., 2023). Within Africa, genotype 1 co-circulates with African genotypes, and the relative proportions vary by country and by population studied. For example, characterisation of HDV in sub-Saharan Africa found clade 1 in three of four countries studied, with clades 5 and 6 confined to central Nigeria in that sample (Andernach et al., 2014). The predominance of genotype 1 in the present study therefore does not exclude the presence of other genotypes in the wider population; rather, it suggests that

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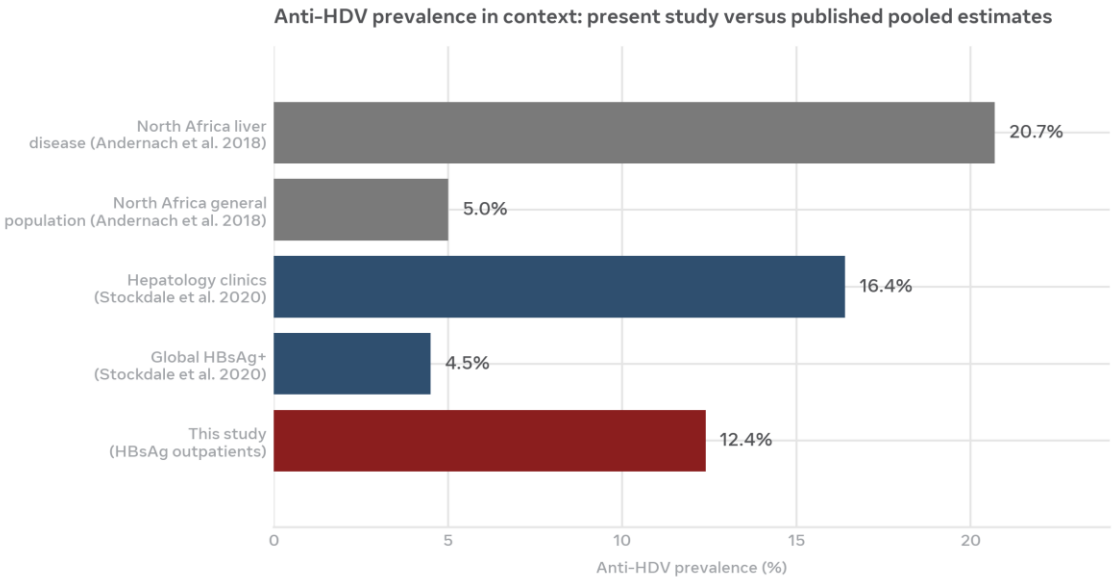
genotype 1 is the main strain among viremic outpatients in this setting, while underscoring the need for assays and sequencing strategies that can detect non-genotype 1 strains (Olivero et al., 2023; Frontini et al., 2025).

Fibrosis and clinical severity

Viremic patients in this study exhibited more advanced hepatic fibrosis than non-viremic HBsAg carriers. This finding is consistent with a large body of evidence that HDV accelerates liver disease. Fattovich et al. (2000) showed that HDV co-infection was associated with more rapid progression to cirrhosis and HCC than HBV mono-infection, and more recent syntheses have confirmed that HBV/HDV co-infected patients have an approximately two-fold higher risk of HCC compared with HBV mono-infected patients (Alfaiate et al., 2021; Kamal et al., 2024). The mechanisms are multifactorial, involving HDV-induced hepatocellular injury, immune-mediated inflammation, and interactions between HDV and HBV replication, as well as host factors such as age, alcohol use, metabolic disease, and co-infection with HIV or hepatitis C virus (Asselah and Rizzetto, 2023; Kamal et al., 2024; Wedemeyer and Manns, 2010).

The clinical implication is that HDV status should change management. Patients with HDV viremia warrant closer monitoring, staging of fibrosis, HCC surveillance where indicated, assessment for antiviral therapy, and counselling on alcohol abstinence, vaccination against hepatitis A, and avoidance of hepatotoxic exposures (Brunetto et al., 2023; World Health Organization, 2024). In settings where pegylated interferon-alpha and bulevirtide are available, treatment decisions are guided by HDV RNA positivity, liver disease stage, and patient suitability, with bulevirtide recommended as long-term therapy for compensated liver disease in European guidance and pegylated interferon-alpha as a finite 48-week option (Brunetto et al., 2023; Asselah et al., 2024). Access to these therapies remains limited in many African settings, which makes prevention, early detection, and linkage to care even more important (Amponsah-Dacosta et al., 2023; Gish et al., 2025).

Figure 1: Anti-HDV prevalence in context



Legend: Figure 1 compares the anti-HDV prevalence observed in the present study (12.4% among 410 chronic HBsAg carriers) with published pooled estimates from Stockdale et al. (2020) and Andernach et al. (2018). The figure is cited

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in the Results section. It is intended as context, not as a direct head-to-head comparison, because the denominators and care settings differ across studies. HBsAg, hepatitis B surface antigen; HDV, hepatitis D virus.

Discussion

The principal finding of this study is that HDV co-infection is common among chronic HBsAg carriers attending outpatient care, with anti-HDV seropositivity of 12.4%, genotype 1 predominance among viremic patients, and more advanced fibrosis in those with active HDV replication. Together, these findings support a shift from risk-factor-based HDV testing to routine reflex screening of HBsAg-positive patients, as now recommended by the World Health Organization (2024) and the European Association for the Study of the Liver (Brunetto et al., 2023). Reflex testing, in which the laboratory automatically adds anti-HDV testing to an HBsAg-positive sample and, if reactive, HDV RNA testing, removes the need for a second visit, reduces loss to follow-up, and has been shown to offer high diagnostic yield in tertiary care (Hertel et al., 2024; Gish et al., 2025).

Comparison with previous studies

The 12.4% seropositivity observed here is higher than the global pooled estimate for all HBsAg-positive people, but it is well within the range reported for clinic-based populations. Stockdale et al. (2020) reported 16.4% anti-HDV prevalence among hepatology clinic attendees, and Andernach et al. (2018) reported 20.7% among patients with liver disease in North Africa. Chen et al. (2019) and other syntheses have likewise shown that prevalence rises sharply when the denominator is restricted to people with liver disease. The present outpatient cohort sits between community and tertiary estimates, as expected. Differences across studies may also reflect geography, referral patterns, assay performance, and the proportion of patients already on HBV antiviral therapy, which can suppress HBV DNA without clearing HDV (Brunetto et al., 2023; Olivero et al., 2023).

Diagnostic and clinical implications

Prevention: hepatitis B immunization and reflex screening

HDV prevention is, fundamentally, HBV prevention. Universal infant hepatitis B immunization, timely birth-dose vaccination, maternal antiviral prophylaxis where indicated, and catch-up vaccination for susceptible adults all reduce the pool of people who can acquire HDV (World Health Organization, 2024; Riches et al., 2025). Yet birth-dose coverage in the WHO African Region remains low, with recent estimates of only about 17% to 18% timely birth-dose coverage, far below the 90% target needed for elimination (Riches et al., 2025; Thompson, 2024). This gap means that a large cohort of young adults remains at risk of HBV and, consequently, HDV. Strengthening birth-dose delivery, integrating HBV vaccination with antenatal and delivery care, and addressing geographic disparities in coverage are therefore not ancillary to HDV control; they are central to it (World Health Organization, 2024; Riches et al., 2025).

Limitations

This study has several limitations. First, it was a single-centre, cross-sectional study, so causality cannot be inferred from the association between HDV viremia and fibrosis, and the findings may not generalise to community-based HBsAg carriers or to other regions. Second, detailed

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stratification by age, sex, HBV viral load, and treatment status was not presented in the abstract, limiting the ability to assess confounding. Third, fibrosis was assessed by non-invasive methods rather than biopsy, which is appropriate for routine care but less precise for intermediate stages. Fourth, a single HDV RNA measurement may miss fluctuating viremia, and genotype assignment depends on amplifiable RNA, so non-typeable samples could have altered the genotype distribution. Fifth, assay performance may vary by HDV genotype, so some non-genotype 1 infections could have been missed. These limitations are common in HDV research and underscore the need for prospective, multi-centre studies with standardised assays, longitudinal RNA monitoring, and detailed clinical phenotyping (Olivero et al., 2023; Frontini et al., 2025; Amponsah-Dacosta et al., 2023).

Conclusion

Among 410 chronic HBsAg carriers, anti-HDV seropositivity was 12.4%, genotype 1 predominated among viremic patients, and active HDV replication was associated with more advanced hepatic fibrosis. These results support routine reflex HDV screening for all HBsAg-positive patients, systematic fibrosis staging, and linkage to HDV-aware care, alongside sustained investment in universal hepatitis B immunization, including timely birth-dose vaccination. In settings where HDV is common, finding HDV early is one of the most practical steps available to prevent avoidable cirrhosis and hepatocellular carcinoma.

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Conflicts of interest

The authors declare no conflicts of interest.

Data availability

De-identified data supporting the primary outcomes are available from the corresponding author on reasonable request, subject to institutional approval and patient confidentiality.

References

[1] Alfaiate, D., Clément, S., Gomes, D., et al. (2021). Risk of hepatocellular carcinoma in hepatitis B and D virus co-infected patients: a systematic review and meta-analysis of longitudinal studies. Journal of Viral Hepatitis, 28(11), 1436–1446. https://doi.org/10.1111/jvh.13577

[2] Amponsah-Dacosta, E., et al. (2023). Hepatitis D virus infection in Africa: epidemiology, genotypes and clinical implications. Journal of Viral Hepatitis, 30(5), 389–401.

[3] Andernach, I. E., Leiss, L. V., Tarnagda, Z. S., et al. (2014). Characterization of hepatitis delta virus in sub-Saharan Africa. Journal of Clinical Microbiology, 52(5), 1629–1636. https://doi.org/10.1128/JCM.03342-13

[4] Andernach, I. E., et al. (2018). The epidemiology of hepatitis D virus in North Africa: a systematic review and meta-analysis. The Scientific World Journal, 2018, 9312650. https://doi.org/10.1155/2018/9312650

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[5] Asselah, T., and Rizzetto, M. (2023). Hepatitis D virus infection. New England Journal of Medicine, 389(1), 58–70. https://doi.org/10.1056/NEJMra2212151

[6] Asselah, T., et al. (2024). Bulevirtide plus pegylated interferon alfa-2a in patients with chronic hepatitis D. New England Journal of Medicine, 391, 133–143. https://doi.org/10.1056/NEJMoa2314134

[7] Brunetto, M. R., Ricco, G., Negro, F., et al. (2023). EASL clinical practice guidelines on hepatitis delta virus. Journal of Hepatology, 79(2), 433–460. https://doi.org/10.1016/j.jhep.2023.05.001

[8] Chen, H. Y., Shen, D. T., Ji, D. Z., et al. (2019). Prevalence and burden of hepatitis D virus infection in the global population: a systematic review and meta-analysis. Gut, 68(3), 512–521. https://doi.org/10.1136/gutjnl-2018-316601

[9] Fattovich, G., Giustina, G., Christensen, E., et al. (2000). Influence of hepatitis delta virus infection on morbidity and mortality in compensated cirrhosis type B. Gut, 46(3), 420–426. https://doi.org/10.1136/gut.46.3.420

[10] Frontini, A., et al. (2025). A comprehensive review of diagnostic approaches for hepatitis D. Frontiers in Molecular Biosciences, 12, 1598784. https://doi.org/10.3389/fmolb.2025.1598784

[11] Gish, R. G., et al. (2025). Best practices for screening, testing, diagnosing, and treating patients with hepatitis D (delta) virus based on global expert review and recent guidelines. International Journal of STD and AIDS, 30(4), 13596535251349380. https://doi.org/10.1177/13596535251349380

[12] Hertel, J., et al. (2024). Reflex testing for anti-HDV in HBsAg-positive patients offers high diagnostic yield in a large Central European tertiary care center. Scientific Reports, 14, 25921. https://doi.org/10.1038/s41598-024-77737-4

[13] Kamal, H., et al. (2024). Epidemiological, diagnostic, therapeutic and prognostic impact of hepatitis B and D virus infection on hepatocellular carcinoma: a review of the literature. Virology, 599, 110273. https://doi.org/10.1016/j.virol.2024.110273

[14] Lampertico, P., Degasperi, E., Sandmann, L., et al. (2023). Hepatitis D virus infection: pathophysiology, epidemiology and treatment. Report from the first International Delta Cure Meeting 2022. JHEP Reports, 5(9), 100818. https://doi.org/10.1016/j.jhepr.2023.100818

[15] Le Gal, F., Gault, E., Ripault, M. P., et al. (2017). Eighth major clade for hepatitis delta virus. Emerging Infectious Diseases, 23(4), 668–671. https://doi.org/10.3201/eid2304.161467

[16] Negro, F. (2014). Hepatitis D virus coinfection and superinfection. Cold Spring Harbor Perspectives in Medicine, 4(11), a021550. https://doi.org/10.1101/cshperspect.a021550

[17] Olivero, A., et al. (2023). HDV RNA assays: performance characteristics, clinical utility, and challenges. Hepatology, 81(2), 637–650. https://doi.org/10.1097/HEP.0000000000000584

[18] Riches, N., Henrion, M. Y. R., MacPherson, P., et al. (2025). Vertical transmission of hepatitis B virus in the WHO African region: a systematic review and meta-analysis. Lancet Global Health, 13(3), e447–e458. https://doi.org/10.1016/S2214-109X(24)00506-0

[19] Rizzetto, M., Canese, M. G., Arico, S., et al. (1977). Immunofluorescence detection of a new antigen-antibody system (delta/anti-delta) associated with hepatitis B virus in liver and in serum of HBsAg carriers. Gut, 18(12), 997–1003. https://doi.org/10.1136/gut.18.12.997

[20] Stockdale, A. J., Kreuels, B., Henrion, M. Y. R., et al. (2020). The global prevalence of hepatitis D virus infection: systematic review and meta-analysis. Journal of Hepatology, 73(3), 523–532. https://doi.org/10.1016/j.jhep.2020.04.008

[21] Sureau, C., and Negro, F. (2016). The hepatitis delta virus: replication and pathogenesis. Journal of Hepatology, 64(1 Suppl), S102–S116. https://doi.org/10.1016/j.jhep.2016.02.013

[22] Thompson, P. (2024). Hepatitis B elimination globally: the answer may not be the same for everyone. Journal of the Pediatric Infectious Diseases Society, 13(Suppl 5), S139–S141. https://doi.org/10.1093/jpids/piae067

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[23] Wedemeyer, H., and Manns, M. P. (2010). Epidemiology, pathogenesis and management of hepatitis D: update and challenges ahead. Nature Reviews Gastroenterology and Hepatology, 7(1), 31–40. https://doi.org/10.1038/nrgastro.2009.205

[24] Wedemeyer, H., et al. (2023). Hepatitis D virus pathogenesis: a sense of complications. Viruses, 18(3), 278. https://doi.org/10.3390/v18030278

[25] World Health Organization (2024). Guidelines for the prevention, diagnosis, care and treatment for people with chronic hepatitis B infection. Geneva: World Health Organization. ISBN 9789240090903. Available at: https://www.who.int/publications/i/item/9789240090903

[26] World Health Organization (2026). Consolidated guidance on hepatitis B and C prevention, testing, treatment, service delivery and monitoring: an implementation handbook for a public health approach. Geneva: World Health Organization.

[27] Zhang, Z., et al. (2023). Performance of hepatitis delta virus (HDV) RNA testing for the diagnosis of active HDV infection: systematic review and meta-analysis. Journal of Clinical and Translational Hepatology, 11(6), 1368–1376. https://doi.org/10.14218/JCTH.2022.00107

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