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Prevalence, Clinicobiochemical, and Immunological Profile of Human Immunodeficiency Virus and Human Immunodeficiency Virus–Hepatitis B Virus Coinfected Patients in a Tertiary Care Hospital in South India

V. C. Srinivas Reddy[1], T. Percy Priyanka[2], B. Jyotirmayi[3]*, Rojalin Mishra[4]

Abstract

Background: Human immunodeficiency virus (HIV) and hepatitis B virus (HBV) share common transmission routes, and coinfection is known to worsen immunological recovery in both conditions; Indian data on the clinicobiochemical profile of HIV–HBV coinfection remain limited. **Methods:** This cross-sectional observational study, conducted at the anti-retroviral therapy (ART) center, Government General Hospital, Vijayawada, over 6 months, aimed to determine the prevalence of HIV–HBV coinfection, compare demographic, clinical, biochemical, and immunological profiles between HIV-monoinfected and HIV–HBV coinfected patients, and assess the association between CD4 T-cell counts and opportunistic infections. Of 1,802 HIV-positive individuals screened for hepatitis B surface antigen, 101 were coinfected (cases), and 100 HIV-monoinfected patients served as controls; data were analyzed using the Statistical Package for the Social Sciences v25. **Results:** The prevalence of HIV–HBV coinfection was 5.6%. Coinfection was more frequent among males (66.3%) and rural residents (73.3%, $P < 0.05$), with heterosexual transmission predominating (82.2%). Coinfected patients had significantly lower mean CD4 counts (214 ± 86 vs. 328 ± 112 cells/µL; $P < 0.001$), more advanced centers for disease control and prevention Stage C disease (50.5% vs. 35.0%), and higher rates of elevated alanine aminotransferase (63.4% vs. 26.0%) and aspartate aminotransferase (74.3% vs. 31.0%; both $P < 0.001$). Opportunistic infections, particularly chronic diarrhea (43.6%) and pulmonary tuberculosis (21.8%), were more common in coinfected patients. **Conclusion:** HIV–HBV coinfection significantly worsens immunosuppression, accelerates hepatic injury, and increases the burden of systemic opportunistic infections. Routine HBV screening in all HIV patients, early initiation of dual-active ART, and targeted rural health interventions are warranted.

Keywords: CD4 count, Coinfection, Hepatitis B virus, Human immunodeficiency virus, Opportunistic infections

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Introduction

Human immunodeficiency virus (HIV) and hepatitis B virus (HBV) remain two of the most significant blood-borne and sexually transmitted pathogens affecting global public health. Because they share transmission routes – sexual contact, percutaneous blood exposure, and perinatal transmission – coinfection with both viruses is common wherever either virus is endemic. According to the World Health Organization's (WHO) Global Hepatitis Report 2026, an estimated 240 million people worldwide were living with chronic HBV infection in 2024, causing approximately 1.1 million deaths annually, predominantly from cirrhosis and hepatocellular carcinoma.[1] Among people living with HIV (PLHIV), HBV coinfection rates of 7–10% have been reported globally, rising to 10–15% in regions of high HBV endemicity such as Sub-Saharan Africa and parts of Asia.[1]

India carries a substantial share of both epidemics. The National Acquired Immunodeficiency Syndrome Control Organization's most recent estimation places the number of PLHIV in the country at approximately 2.5 million.[2] Independently, an estimated 40 million Indians live with chronic HBV infection.[3] This overlapping burden creates a large population at risk of dual infection, with important clinical consequences: HIV-associated immunosuppression accelerates HBV replication and hastens progression of liver fibrosis (FIB), while HBV coinfection blunts CD4 T-cell recovery and worsens overall HIV-related morbidity.

Despite the scale of this overlap, India-specific data describing the detailed clinical, biochemical, and immunological consequences of HIV–HBV coinfection remain sparse and geographically uneven, drawn largely from cohorts in North, East, and South India studied more than a decade ago.[4-6] More recent regional data, including a 2024 study from a neighboring anti-retroviral therapy (ART) center in Eluru, Andhra Pradesh,[7] and molecular characterization work from North India,[3] indicate that the epidemiology and genetic diversity of coinfecting HBV strains continue to evolve, reinforcing the need for updated, locally generated evidence.

[1]Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India.

[2]Associate Professor, Department of General Medicine, Dr. Yellapragada Subba Rao Medical College, Eluru, Andhra Pradesh, India.

[3]Associate Professor, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India.

[4]Junior Resident, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India.

Corresponding Author: B. Jyotirmayi, Department of General Medicine, Government Siddhartha Medical College, Vijayawada, Andhra Pradesh, India. E-mail: drboddudgo2001@yahoo.com

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The pathophysiological interaction between the two viruses is bidirectional and clinically consequential. HIV-induced depletion of CD4 T-helper lymphocytes and B-cell dysfunction impair the host's capacity to mount an effective anti-HBV cytotoxic T-cell and antibody response, permitting higher and more sustained HBV viremia, reduced rates of spontaneous hepatitis B e antigen (HBeAg) seroconversion, and accelerated progression to cirrhosis and hepatocellular carcinoma relative to HBV monoinfection.[8] Conversely, chronic HBV replication contributes to persistent hepatic and systemic immune activation, which is thought to blunt CD4 T-cell reconstitution following initiation of ART and to be associated with a higher incidence of acquired immunodeficiency syndrome (AIDS)-defining illnesses.[9] Hepatotoxicity related to certain antiretroviral agents, and the phenomenon of immune reconstitution inflammatory syndrome affecting the liver after ART initiation, add further clinical complexity to the management of coinfected patients and underscore the need for close biochemical and virological surveillance in this group.

Andhra Pradesh has historically been among the states with the highest reported HIV burden in India, and Vijayawada serves as a referral hub for a large rural and semi-urban catchment population in the Krishna River basin.[2] Given the state's substantial HIV caseload, the persistence of low HBV vaccination coverage among adults born before the introduction of universal infant HBV immunization, and the scarcity of contemporary regional data, a tertiary-care ART center in this setting offers a valuable vantage point from which to characterize the current burden and clinical impact of HIV–HBV coinfection. The present study was therefore undertaken with three specific objectives: (i) to determine the prevalence of HIV–HBV coinfection among HIV-positive individuals attending the ART center; (ii) to compare the demographic, clinical, biochemical, and immunological profiles of HIV-monoinfected and HIV–HBV coinfected patients; and (iii) to assess the association between CD4 T-cell counts and the pattern of opportunistic infections in the two groups.

Materials and Methods

Study Design and Setting

This was a hospital-based, cross-sectional observational study conducted at the ART Centre, New Government General Hospital, Vijayawada, Andhra Pradesh, over a 6-month period.

Ethical Considerations

The study protocol was approved by the Institutional Ethics Committee, Government Siddhartha Medical College, Vijayawada (Approval No. IECSMCGGH/2024/AP/034). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment, and confidentiality of HIV/HBV status was maintained throughout using anonymized study identifiers.

Study Population and Sample Size

The study population comprised all HIV-positive adults (age 18–60 years) attending the ART center during the study period. The minimum required sample size was calculated using the standard formula for estimating a single proportion, $n = Z^2P(1-P)/d^2$, assuming an expected HIV–HBV coinfection prevalence (P) of 6% based on prior North Indian data,[4] a 95% confidence level (Z = 1.96), and an absolute precision (d) of 6%, yielding a minimum requirement of approximately 60 coinfected cases. A total of 1,802 HIV-positive individuals were screened for hepatitis B surface antigen (HBsAg) during the study period using a consecutive sampling approach, exceeding the minimum required sample size.

Study Groups

Cases (Coinfected Group): HIV-positive and HBsAg-positive patients ($n = 101$).

Controls (Mono-infected Group): HIV-positive and HBsAg-negative patients ($n = 100$), recruited consecutively from among screened HIV-positive attendees during the same period.

Eligibility Criteria

Inclusion criteria

(1) Confirmed HIV infection by National AIDS Control Organization (NACO)-approved serological algorithm; (2) age 18–60 years; and (3) willingness to provide informed consent and comply with study protocols.

Exclusion criteria

(1) Indeterminate HIV test results; (2) prior HBV vaccination; (3) pregnancy; and (4) inability or unwillingness to participate.

Diagnostic Investigations

All participants underwent standardized diagnostic evaluation, comprising: Hematology and Serology (hemoglobin [Hb], total leukocyte count, differential leukocyte count, erythrocyte sedimentation rate, HBsAg screening by rapid immunochromatography with enzyme-linked immunosorbent assay (ELISA) confirmation, and HIV confirmatory testing per NACO protocol); Biochemistry (blood urea, serum creatinine, and liver function tests including alanine aminotransferase [ALT] and aspartate aminotransferase [AST]); immunology and virology (absolute CD4 T-cell counts by flow cytometry, and quantitative HBV DNA polymerase chain reaction for all coinfected cases); Opportunistic Infection Screening (sputum smear microscopy/cartridge-based nucleic acid amplification test for acid-fast bacilli, stool microscopy for ova and cysts, urine microscopy, and cerebrospinal fluid analysis where clinically indicated); and radiological imaging (chest radiography, high-resolution computed tomography of the chest, and abdominal ultrasonography, as clinically indicated).

Clinical Staging and Case Definitions

Clinical staging of HIV disease was performed according to the centers for disease control and prevention (CDC) 1993 classification system, categorizing patients into Stage A (asymptomatic or acute primary infection), Stage B (symptomatic, non-AIDS-defining conditions), and Stage C (AIDS-defining conditions).

HBsAg-positive patients were further evaluated for HBeAg status and quantitative HBV DNA viral load, with active viral replication defined as HBV DNA >2,000 IU/mL, consistent with commonly used treatment-eligibility thresholds for chronic hepatitis B. Anemia was defined as Hb <10 g/dL, and elevated transaminases were defined as ALT or AST >40 U/L, in line with standard laboratory reference ranges.

Data Quality and Bias Control

To minimize measurement and selection bias, all HBsAg screening was performed using a standardized rapid immunochromatography assay with mandatory ELISA confirmation of reactive samples, and CD4 enumeration was performed on a single calibrated flow cytometer at the center's designated CD4 laboratory. Cases and controls were recruited concurrently from the same screening pipeline, using the same eligibility criteria and the same trained data-collection team, to reduce the likelihood of temporal or observer-related bias between groups. Data were cross-checked against ART center registers by a second investigator prior to entry into the statistical software.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using IBM Statistical Package for the Social Sciences Statistics version 25. Categorical variables were expressed as frequencies and percentages and compared between the coinfected and mono-infected groups using the Chi-square test, or Fisher's exact test where expected cell counts were below five. Continuous variables (age, CD4 count) were expressed as mean ± standard deviation and compared using the independent-samples Student's *t*-test after visual assessment of approximate normality. A two-tailed $P < 0.05$ was considered statistically significant throughout. No formal adjustment for multiple comparisons was applied, given the primary descriptive and hypothesis-generating intent of the study; this is discussed further as a limitation.

Results

Prevalence of HIV–HBV Coinfection

Of 1,802 HIV-positive patients screened at the ART center during the study period, 101 were confirmed positive for HBsAg, yielding an overall HIV–HBV coinfection prevalence of 5.6%.

Demographic Profile

A comparison of demographic characteristics between HIV–HBV coinfected cases ($n = 101$) and HIV mono-infected controls ($n = 100$) is summarized in Tables 1-4. Coinfection was more frequent among males (66.3% vs. 62.0%) and married patients (67.3% vs. 65.0%), though neither difference reached statistical significance [Tables 1 and 2].

Table 1: Gender distribution among coinfected and mono-infected groups

Gender	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Male	67 (66.3)	62 (62.0)	>0.05
Female	34 (33.7)	38 (38.0)	—

The age distribution was broadly similar between the two groups, with the majority of patients in both belonging to the 21–40 year bracket (61.2% overall, 123/201), representing the sexually active and economically productive segment of the population; only 5.0% of the overall cohort was aged 20 years or younger, and coinfection was not observed in any patient older than 60 years [Table 3]. Illiteracy (67.3% vs. 60.0%) and rural residence (73.3% vs. 60.0%, $P < 0.05$) were both more common among coinfected patients, with rural residence reaching statistical significance [Table 4] – an observation explored further in the discussion.

Modes of Transmission

Heterosexual contact was the predominant route of infection in both groups (82.2% in cases vs. 86.0% in controls), followed by blood transfusion (6.9% vs. 4.0%), injecting drug use (5.0% vs. 3.0%), and unknown or undisclosed routes (5.9% vs. 7.0%) [Table 5]. None of the between-group differences in transmission category reached statistical significance, suggesting that in this population, the risk of acquiring HBV coinfection is distributed across the same behavioral and exposure categories that drive HIV transmission itself, rather than being concentrated in a distinct high-risk subgroup.

Clinical Presentation

Coinfected patients demonstrated a significantly higher symptom burden than mono-infected controls, including fever (81.2% vs. 70.0%, $P < 0.05$), general weakness (71.3% vs. 26.0%, $P < 0.001$), diarrhea (43.6% vs. 13.0%, $P < 0.001$), and vomiting (20.8% vs. 8.0%,

Table 2: Marital status distribution

Marital status	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Married	68 (67.3)	65 (65.0)	>0.05
Unmarried	19 (18.8)	22 (22.0)	>0.05
Widowed/Divorced	14 (13.9)	13 (13.0)	>0.05

Table 3: Age group distribution

Age group (years)	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*Total (n=201) (%)*
2–20	4 (4.0)	6 (6.0)	10 (5.0)
21–40	65 (64.4)	58 (58.0)	123 (61.2)
41–60	32 (31.7)	32 (32.0)	64 (31.8)
>60	0 (0.0)	4 (4.0)	4 (2.0)

The majority of patients belonged to the 21–40 years age bracket (61.2%), representing the sexually active and economically productive segment

Table 4: Literacy and residential background

Parameter	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Illiterate	68 (67.3)	60 (60.0)	>0.05
Rural Residence	74 (73.3)	60 (60.0)	<0.05*

*Statistically significant difference ($P < 0.05$)

Table 5: Mode of transmission

Mode of transmission	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*
Heterosexual	83 (82.2)	86 (86.0)
Blood transfusion	7 (6.9)	4 (4.0)
Injecting drug use	5 (5.0)	3 (3.0)
Unknown/undisclosed	6 (5.9)	7 (7.0)

P < 0.05). Cough and asymptomatic presentation were, conversely, more common among controls [Table 6].

CDC Clinical Staging

Advanced immunodeficiency (CDC Stage C) was significantly more common among coinfected subjects (50.5% vs. 35.0%, *P* < 0.05), while Stage A (asymptomatic/primary) was more frequent among controls (24.0% vs. 10.9%, *P* < 0.05) [Table 7]. Stage B disease was similarly distributed between the two groups (38.6% vs. 41.0%), indicating that the principal divergence between cases and controls lay at the extremes of disease severity rather than in the intermediate, symptomatic-but-non-AIDS-defining category.

Immunological Profile (CD4 T-Cell Count)

Coinfected patients had a markedly lower mean CD4 count than mono-infected patients (214 ± 86 vs. 328 ± 112 cells/μL; *P* < 0.001). A CD4 count below 200 cells/μL was seen in 50.5% of coinfected patients compared with 35.0% of controls (*P* < 0.05) [Table 8].

Virological Profile (HBV DNA)

Among the 101 HIV–HBV coinfected patients, quantitative HBV DNA analysis revealed active replication (>2,000 IU/mL) in 62 patients (61.4%), while 39 patients (38.6%) had HBV DNA below this threshold. The predominance of high-level replication in this cohort is consistent with impaired HBV-specific immune control in the setting of HIV-associated immunosuppression and identifies a majority of coinfected patients as candidates for structured antiviral treatment and monitoring under current national and international hepatitis B guidelines.

Table 6: Clinical symptoms profile

Symptom	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Fever	82 (81.2)	70 (70.0)	<0.05*
General weakness	72 (71.3)	26 (26.0)	<0.001*
Weight loss	64 (63.4)	56 (56.0)	>0.05
Diarrhea	44 (43.6)	13 (13.0)	<0.001*
Cough	29 (28.7)	43 (43.0)	<0.05*
Vomiting	21 (20.8)	8 (8.0)	<0.05*
Asymptomatic	11 (10.9)	24 (24.0)	<0.05*

*Statistically significant difference (*P* < 0.05)

Table 7: Distribution by CDC clinical stage

CDC stage	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Stage A (Asymptomatic/Primary)	11 (10.9)	24 (24.0)	<0.05*
Stage B (Symptomatic Non-A/C)	39 (38.6)	41 (41.0)	>0.05
Stage C (AIDS-defining)	51 (50.5)	35 (35.0)	<0.05*

*Statistically significant difference (*P* < 0.05)

Table 8: CD4 T-cell count distribution

CD4 count (cells/μL)	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
<200	51 (50.5)	35 (35.0)	<0.05*
200–499	39 (38.6)	41 (41.0)	>0.05
>500	11 (10.9)	24 (24.0)	<0.05*
Mean CD4±standard deviation	214±86	328±112	<0.001*

*Statistically significant difference (*P* < 0.05)

Biochemical and Hematological Profile

Hepatic enzyme elevation and anemia were significantly more frequent in coinfected patients. Elevated ALT (>40 U/L) was seen in 63.4% of coinfected patients versus 26.0% of controls, and elevated AST (>40 U/L) in 74.3% versus 31.0% (both *P* < 0.001). Anemia (Hb <10 g/dL) was also significantly more common among coinfected patients (67.3% vs. 52.0%, *P* < 0.05) [Table 9].

Opportunistic Infections

Systemic opportunistic infections – specifically chronic diarrhea (43.6% vs. 13.0%, *P* < 0.001) and pulmonary tuberculosis (21.8% vs. 11.0%, *P* < 0.05) – occurred at significantly higher rates in coinfected patients, whereas oral candidiasis was more common among controls (41.0% vs. 18.8%, *P* < 0.001) [Table 10].

Discussion

In this hospital-based cross-sectional study conducted at a tertiary-care ART center in Vijayawada, Andhra Pradesh, the prevalence of HIV–HBV coinfection was 5.6%. This finding is consistent with the intermediate endemicity typically reported from Indian cohorts, which generally range between 4% and 10% depending on region and testing methodology.[4-6] It is closely comparable to a contemporaneous 2024 study from a neighboring ART center in Eluru, Andhra Pradesh, in which HIV-positive patients on ART showed significantly lower baseline CD4 counts when coinfected with HBV compared with HIV-monoinfected patients (223 vs. 310 cells/μL)[7] – a pattern that mirrors, albeit at somewhat higher absolute values, the pronounced CD4 deficit observed in the present cohort (214 vs. 328 cells/μL). A recent molecular epidemiology study from North India similarly reported active HBV replication and a substantial burden of HBsAg immune-escape mutations among coinfected patients,[3] reinforcing the clinical relevance of virological monitoring in this population.

Demographically, coinfection predominated among males (66.3%) and patients residing in rural regions (73.3%, *P* < 0.05), consistent with the "ruralization" of the HIV epidemic increasingly described in Indian surveillance data.[2] Lower educational attainment, limited awareness of viral hepatitis transmission, and the absence of universal childhood HBV vaccination in older birth

Table 9: Hematological and biochemical abnormalities

Parameter	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Anemia (Hb <10 g/dL)	68 (67.3)	52 (52.0)	<0.05*
Elevated ALT (>40 U/L)	64 (63.4)	26 (26.0)	<0.001*
Elevated AST (>40 U/L)	75 (74.3)	31 (31.0)	<0.001*
Elevated urea (>40 mg/dL)	12 (11.9)	8 (8.0)	>0.05
Elevated serum creatinine (>1.4 mg/dL)	8 (7.9)	5 (5.0)	>0.05

*Statistically significant difference (*P* < 0.05). Hb: Hemoglobin, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase

Table 10: Spectrum of opportunistic infections

Opportunistic infection	*Coinfected (n=101) (%)*	*Mono-infected (n=100) (%)*	*P-value*
Chronic diarrhea	44 (43.6)	13 (13.0)	<0.001*
Pulmonary tuberculosis	22 (21.8)	11 (11.0)	<0.05*
Oral candidiasis	19 (18.8)	41 (41.0)	<0.001*
No opportunistic infection	14 (13.9)	34 (34.0	<0.01*

*Statistically significant difference (*P*<0.05)

cohorts likely contribute to this rural clustering – a pattern also highlighted in a Central Indian hospital-based hepatitis screening study that found higher HBV-related seropositivity among under-served, lower-literacy populations.[10]

Clinically, HIV–HBV coinfected patients in the present study exhibited a distinctly more severe phenotype than mono-infected controls. Over half (50.5%) of coinfected individuals presented in CDC Stage C (AIDS-defining illness), compared with 35.0% of controls, and mean CD4 counts were significantly lower in the coinfected group (214 ± 86 vs. 328 ± 112 cells/μL; $P < 0.001$). These findings align with the broader literature indicating that HBV coinfection independently blunts CD4 T-cell recovery in PLHIV, potentially through chronic immune activation, altered cytokine signaling, and direct hepatic viral interactions.[8,9] A comparable pattern was reported in a 2024 Ethiopian ART-clinic cohort, in which HBV/HCV coinfected patients showed poorer immunological outcomes than HIV-monoinfected peers, underscoring that this immunological penalty of coinfection is not confined to the Indian subcontinent.[11]

Biochemically, elevated ALT and AST were substantially more frequent among coinfected patients (63.4% and 74.3%, respectively) than controls (26.0% and 31.0%; both $P < 0.001$), and 61.4% of coinfected patients showed active HBV replication (HBV DNA >2,000 IU/mL). This is consistent with the well-established mechanism by which HIV-related impairment of T-cell-mediated viral clearance permits uninhibited HBV replication, accelerating hepatocyte injury, FIB progression, and long-term risk of cirrhosis and hepatocellular carcinoma.[12] A 2025 Romanian cohort study similarly demonstrated that HBV and/or HCV coinfection independently predicted abnormal liver enzyme trajectories and blunted virological response to ART in HIV-infected patients, reinforcing the case for routine, structured hepatic monitoring in all coinfected individuals on ART, irrespective of geography.[13]

The spectrum of opportunistic infections also differed markedly between groups. Coinfected patients experienced substantially higher rates of chronic diarrhea (43.6% vs. 13.0%) and pulmonary tuberculosis (21.8% vs. 11.0%), consistent with advanced gut-associated lymphoid tissue depletion and blunted cell-mediated immunity in the setting of dual viral suppression of host defences. Conversely, oral candidiasis was more frequent in mono-infected controls (41.0% vs. 18.8%), possibly reflecting differences in baseline ART exposure duration, local mucosal immune factors, or detection bias related to more frequent clinical examination of visibly symptomatic patients.

Taken together, these findings reinforce current international and national guidance. The WHO's 2024 hepatitis B guidelines emphasise simplified, expanded HBV testing and treatment eligibility, including in populations coinfected with HIV, and recommend structured monitoring for hepatitis delta coinfection where relevant.[14] Current NACO ART guidelines already recommend tenofovir-based, dual-active regimens for coinfected patients;[15] the findings of the present study support the continued prioritization of this approach, alongside earlier and more systematic HBsAg screening at ART initiation, particularly in rural and lower-literacy populations who appear to bear a disproportionate share of the coinfection burden in this region.

From a health-systems perspective, these findings carry direct policy relevance. HBsAg testing at ART centers in India is not yet universally mandated at the point of HIV diagnosis, and where it is performed, follow-up HBV DNA quantification and FIB staging are often unavailable outside larger tertiary centers. Given that nearly two-thirds of coinfected patients in this cohort had active HBV replication and correspondingly elevated transaminases, the marginal cost of incorporating routine HBsAg screening and periodic liver-function monitoring into existing ART follow-up protocols is likely to be justified by the reduction in missed or delayed diagnoses of clinically significant liver disease. Because tenofovir-based ART regimens already provide dual anti-HIV and anti-HBV activity, the additional resource requirement for improving coinfection outcomes lies chiefly in diagnostics and monitoring rather than in a fundamentally different treatment strategy,[14,15] making this an achievable near-term target for program strengthening within the National AIDS Control Programme.

An additional consideration, not directly assessed in this study but increasingly emphasised in updated international guidance, is the role of hepatitis D virus (HDV) in patients coinfected with HIV and HBV. The 2024 WHO hepatitis B guidelines recommend reflex HDV serological testing in HBsAg-positive individuals from settings of intermediate-to-high HDV prevalence[14], and emerging cohort data suggest that HDV coinfection, where present, can further worsen liver-related outcomes in HIV–HBV coinfected patients. HDV status was not evaluated in the present cohort; incorporating HDV screening into future iterations of this ART center's coinfection protocol, particularly in patients with unexpectedly aggressive liver enzyme elevation, would help to characterize the full spectrum of viral hepatitis burden in this population. Similarly, the near-universal absence of documented adult HBV vaccination among enrolled patients (a prerequisite exclusion criterion notwithstanding) reflects a broader gap in catch-up hepatitis B immunization for adults born before India's introduction of universal infant HBV vaccination in the early 2000s – a gap that targeted rural outreach and ART-center-based vaccination drives could help to close over time.

Future Research Directions

Future work at this center and comparable ART settings in Andhra Pradesh should prioritize prospective, longitudinal follow-up of coinfected patients through at least the first 12–24 months of tenofovir-based ART, to characterize the trajectory of CD4 recovery, HBV DNA suppression, and liver-enzyme normalization under treatment. HBV genotyping and surface-gene mutation analysis, as performed in recent North Indian cohorts, would help determine whether locally circulating HBV strains carry immune-escape or drug-resistance-associated mutations relevant to vaccine and treatment planning. Non-invasive FIB staging using AST to platelet ratio index (APRI) or FIB-4 scores at baseline and during follow-up would also allow this cohort to be risk-stratified for early hepatology referral.

Strengths

The principal strengths of this study include its relatively large sample of confirmed coinfected patients drawn from a high-volume ART center, the use of a well-matched HIV-monoinfected comparison group screened during the same period, and the comprehensive diagnostic battery encompassing clinical staging, quantitative CD4 and HBV DNA measurement, hematological and biochemical profiling, and structured opportunistic-infection

screening. This breadth allowed multiple domains of the coinfection phenotype – demographic, behavioral, immunological, virological, and biochemical – to be examined within a single, internally consistent cohort, rather than relying on previously published, geographically disparate data collected under differing protocols and assay platforms.

Limitations

This study has several limitations. Its cross-sectional design precludes causal inference regarding the temporal relationship between coinfection and immunological decline. The single-center setting may limit generalisability to other regions of India. HBV genotyping and resistance-mutation analysis, which have been shown elsewhere to influence treatment response and immune escape,[3] were not performed in this cohort. Finally, potential confounders such as ART regimen duration, adherence, and alcohol use were not systematically captured and could not be adjusted for in the present analysis.

Conclusion

The prevalence of HIV–HBV coinfection at this tertiary-care center in Vijayawada was 5.6%, predominantly affecting young, rural males through heterosexual transmission. HIV–HBV coinfection represents a distinct and more severe clinical entity, characterised by profound immunosuppression, higher CDC clinical staging, pronounced liver enzyme elevation, and an increased burden of systemic opportunistic infections such as tuberculosis and chronic diarrhea, compared with HIV monoinfection.

Clinical and Public Health Recommendations

1. Universal HBV screening: Mandatory baseline screening for HBsAg should be performed in all newly diagnosed HIV patients before ART initiation, given the substantial prevalence (5.6%) and largely silent early course of coinfection observed in this cohort.
2. Dual-active antiretroviral regimens: First-line ART regimens containing tenofovir (tenofovir disoproxil fumarate or tenofovir alafenamide) combined with lamivudine (3TC) or emtricitabine (FTC) should be routinely prescribed in coinfected patients to suppress both viruses simultaneously and to reduce the risk of selecting resistant HBV mutants that could otherwise emerge under HBV-inactive regimens.
3. Hepatic monitoring: Regular monitoring of liver function tests (ALT/AST), periodic HBV DNA quantification, and non-invasive FIB assessment (APRI/FIB-4 or FibroScan, where available) should be instituted for coinfected individuals, given the high prevalence of active viral replication and transaminase elevation documented in this study.
4. Targeted rural health interventions: Community-level awareness campaigns, decentralized screening drives, and catch-up HBV vaccination programs should be prioritized for rural populations, who bore a disproportionate share of the coinfection burden in this cohort.
5. Reflex HDV testing: In line with current WHO guidance, reflex hepatitis D serological testing should be considered for HBsAg-positive patients, particularly those with disproportionately severe liver enzyme derangement relative to HBV DNA levels.

Conflicts of Interest

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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References

1. World Health Organization. Global Hepatitis Report 2026. Geneva: World Health Organization; 2026.
2. National AIDS Control Organization. HIV Estimation 2025 Technical Report. New Delhi: Ministry of Health and Family Welfare, Government of India; 2025.
3. Sami H, Asaad M, Husaini SH, Khan PA, Raza A, Fatima N, *et al*. HBV genotype distribution and S gene mutations in HIV-HBV co-infected patients: Insights from North India. Front Cell Infect Microbiol 2026;15:1731472.
4. Tripathi AK, Khanna M, Gupta N, Chandra M. Low CD4 count and risk of hepatitis B co-infection in HIV-positive patients in North India. J Assoc Physicians India 2007;55:429-31.
5. Saha MK, Chakrabarti S, Panda S, Naik TN, Manna B, Chatterjee A, *et al*. Prevalence of HCV & HBV infection amongst HIV seropositive intravenous drug users and their non-injecting wives in Manipur, India. Indian J Med Res 2000;111:37-9.
6. Saravanan S, Velu V, Kumarasamy N, Nandakumar S, Murugavel KG, Balakrishnan P, *et al*. Coinfection of hepatitis B and hepatitis C virus in HIV-infected patients in south India. World J Gastroenterol 2007;13:5015-20.
7. Manjula V, Krishnaveni A, Viswabharathi N, Jayalakshmi L, Sasidhar M, Jyothirmai M. Prevalence of co-infection of hepatitis B virus and hepatitis C virus among human immunodeficiency virus patients on antiretroviral therapy at a tertiary care centre, Eluru, Andhra Pradesh, India. J Pure Appl Microbiol 2024;18:2328-35.
8. Thio CL. Hepatitis B and human immunodeficiency virus coinfection. Hepatology 2009;49 Suppl 5:S138-45.
9. Colin JF, Cazals-Hatem D, Loriot MA, Martinot-Peignoux M, Pham BN, Auperin A, *et al*. Influence of HIV infection on chronic hepatitis B in hybrid capture positive patients. Hepatology 1999;29:1306-10.
10. Jain RK, Shrivastava R, Jain SK, Chaurasia D, Jain A, Jain S, *et al*. Seropositivity and coinfection of hepatitis B and hepatitis C viruses in Central India: A hospital-based study. J Family Med Prim Care 2024;13:4413-8.
11. Hagos YM, Yalew GT, Meles HN, Tsegay E, Lemelem M, Wasihun AG. Hepatitis B and C viral coinfection and associated factors among HIV-positive patients attending ART clinics of Afar regional state, Northeast Ethiopia. PLoS One 2024;19:e0302453.
12. Barth RE, Huijgen Q, Taljaard J, Hoepelman AI. Hepatitis B/C and HIV in sub-Saharan Africa: An association between highly prevalent infectious diseases. Expert Rev Anti Infect Ther 2010;8:1021-31.
13. Marin RC, Tit DM, Bungău G, Moleriu RD. The impact of hepatitis B and/or C on liver function and on the response to antiretroviral therapy in HIV-infected patients: A Romanian cohort study. Pharmaceuticals (Basel) 2025;18:688.
14. World Health Organization. Guidelines for the Prevention, Diagnosis, Care and Treatment for People with Chronic Hepatitis B Infection. Geneva: World Health Organization; 2024.
15. National AIDS Control Organisation (NACO). National Technical Guidelines for Anti-Retroviral Therapy. New Delhi: Ministry of Health and Family Welfare, Government of India; 2021.